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Handbook of Anesthesiology

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Cardiopulmonary Resuscitation and ACLS

Universal Algorithm for Adult Emergency Cardiac Care

1. Assess responsiveness.
2. Activate EMS (emergency medical services) system.
3. Call for defibrillator.
4. Assess breathing (open the airway, look, listen, and feel).
    A. If the patient is breathing, place in the rescue or recovery position if no trauma is apparent.
    B. If the patient is not breathing normally, give two slow breaths.
5. Assess circulation
    A. If there is a pulse, attempt to categorize the patient according to one of the following suspected causes, and then go to the appropriate algorithm.
        1. Hypotension; shock; acute pulmonary edema.
        2. Acute myocardial infarction.
        3. Arrhythmia that is either too fast or too slow.
    B. If there is no pulse
        1. Start CPR and check whether ventricular fibrillation (VF) or ventricular tachycardia (VT) is present on the monitor/defibrillator.
        2. If VF/VT is present, go to the VF/VT algorithm.
        3. If there is no VF/VT, intubate, confirm tube placement, confirm ventilations, determine rhythm and cause.
    C. If there is electrical activity with no pulse, go to the pulseless activity algorithm.

V-Fibrillation/Pulseless V-Tachycardia Algorithm (VF/VT)

1. Airway, breathing, and circulation (ABCs).
2. CPR until defibrillator available.
3. Defibrillate.
    A. 200 J (unsynchronized).
    B. 200-300 J (unsynchronized).
    C. 360 J (unsynchronized).
4. CPR, IV, intubate. Continue if persistent or recurrent VF/VT.
5. Epinephrine, 1:10,000, 1.0 mg IVP, repeat every 3-5 minutes.
    A. Alternative epinephrine dosing
        1. Intermediate dose: epinephrine 2-5 mg IVP every 3-5 minutes.
        2. Escalating dose: epinephrine 1 mg, 3 mg, and 5 mg IVP given 3 minutes apart.
        3. High dose: epinephrine 0.1 mg/kg IVP, repeat every 3-5 minutes.
6. Defibrillate 360 J (unsynchronized) within 30-60 seconds. Multiple sequenced shocks are acceptable here (Class I), especially when medications are delayed.
7. Lidocaine 1.5 mg/kg IVP, repeat every 3-5 minutes to a total loading dose of 3 mg/kg; then use.
    A. Bretylium, 5 mg/kg IV, repeat with 10 mg/kg every 15-30 minutes to total of 30 mg/kg.
    B. Magnesium sulfate 1-2 grams IV in Torsades de Pointes or suspected hypomagnesemic state or severe refractory VF.

   C. Procainamide 30 mg/min in refractory ventricular fibrillation (maximum total 17 mg/kg).
8. Defibrillate 360 J, 30-60 seconds after each dose of medications. Pattern should be drug-shock, drug-shock.
9. Consider bicarbonate 1 meq/kg (if known preexisting bicarbonate responsive acidosis, overdose with tricyclic antidepressant.

V-Fibrillation/Pulseless V-Tachycardia Algorithm (VF/VT)
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   B. Magnesium sulfate 1-2 grams IV in Torsades de Pointes or suspected hypomagnesemic state or severe refractory VF.
   C. Procainamide 30 mg/min in refractory ventricular fibrillation (maximum total 17 mg/kg).
8. Defibrillate 360 J, 30-60 seconds after each dose of medications. Pattern should be drug-shock, drug-shock.
9. Consider bicarbonate 1 meq/kg (if known preexisting bicarbonate responsive acidosis, overdose with tricyclic antidepressants, prolonged arrest, hypoxic lactic acidosis).

Asystole Algorithm
1. Airway, breathing, and circulation (ABCs)**.**
2. CPR, Intubate, start IV.
3. Confirm asystole in two or more leads.
4. Consider possible causes: hypoxia, hyperkalemia, hypokalemia, preexisting acidosis, drug overdose, and hypothermia.
5. Consider immediate transcutaneous pacing (usually not routine).
6. Epinephrine, 1:10,000, 1.0 mg IVP, repeat every 3-5 minutes.
   A. Alternative epinephrine dosing (Class IIb dosing regimens)
      1. Intermediate dose: epinephrine 2-5 mg IVP every 3-5 minutes.
      2. Escalating dose: epinephrine 1 mg, 3 mg, and 5 mg IVP given 3 minutes apart.
      3. High dose: epinephrine 0.1 mg/kg IVP, repeat every 3-5 minutes.

7. Atropine 1.0 mg IV, repeat every 3-5 minutes to total of 0.04 mg/kg.
8. Consider bicarbonate 1 meq/kg (if known preexisting bicarbonate responsive acidosis, overdose with tricyclic antidepressants, prolonged arrest, hypoxic lactic acidosis).
9. Reconsider pacemaker. Transcutaneous pacing is a Class IIb intervention. Lack of success may be due to delays in pacing. To be effective transcutaneous pacing must be preformed early, simultaneously with drugs. Evidence does not support routine use of transcutaneous pacing for asystole.

PuIseless Electrical Activity Algorithm (Electromechanical dissociation)

1. PuIseless electrical activity includes: EMD, pseudo-EMD, idio-ventricular rhythms, ventricular escape rhythms, postdefibrillation idioventricular rhythms.
2. Airway, breathing, and circulation (ABCs)**.**
3. CPR.
4. Intubate, start IV.
5. Consider possible causes: pericardial tamponade, tension pneumothorax, hypovolemia, massive pulmonary embolus, hypoxia, hypothermia, drug overdose (such as tricyclics, digitalis, beta blockers, calcium channel blockers), hyperkalemia, acidosis, massive acute myocardial infarction.
6. Epinephrine, 1:10,000, 1.0 mg IVP, repeat every 3-5 minutes.
   A. Alternative epinephrine dosing (Class IIb dosing regimens)
      1. Intermediate dose: epinephrine 2-5 mg IVP every 3-5 minutes.
      2. Escalating dose: epinephrine 1 mg, 3 mg, and 5 mg IVP given 3 minutes apart.
      3. High dose: epinephrine 0.1 mg/kg IVP, repeat every 3-5 minutes.
7. If absolute bradycardia (<60 bpm) or relative bradycardia, give atropine 1 mg IVP every 3-5 minutes up to a total of 0.04 mg/kg.
8. Consider bicarbonate 1 mEq/kg (if known preexisting bicarbonate responsive acidosis, overdose with tricyclic antidepressants, prolonged arrest, hypoxic lactic acidosis).

Bradycardia Algorithm

1. Airway, breathing, and circulation (ABCs).
2. Administer oxygen; start IV.
3. Assess vital signs (place all monitors).
4. If time allows review history, examine pt, order 12 lead EKG, portable CXR.
5. If unstable (considered unstable if chest pain, shortness of breath, decreased level of consciousness, hypotension, shock, pulmonary congestion, congested heart failure, or acute myocardial infarction are present).
   A. Atropine 0.5-1.0 mg IVP repeated every 3-5 minutes up to 0.04 mg/kg (note: denervated transplanted hearts will not respond to atropine, go immediately to transcutaneous cardiac pacing).
   B. Transvenous pacing: if patient is symptomatic do not delay transcutaneous cardiac pacing while awaiting IV access or for atropine to take effect .
   C. Dopamine 5-20 mcg/kg/min.
   D. Epinephrine 2-20 mcg/min.
   E. Isoproterenol 0.5-20 mcg/min .
6. If stable and not in type II or type III AV heart block, observe.

7. If type II or type III AV heart block prepare for transvenous pacer (never treat third-degree heart block plus ventricular escape beats with lidocaine).

Tachycardia Algorithm

1. Airway, breathing, and circulation (ABCs).
2. Administer oxygen; secure airway; Start IV.
3. Sssess vital signs (place all monitors).
4. If time allows review history, examine pt, order 12 lead EKG, portable CXR.
5. If unstable (considered unstable if chest pain, hypotension, CHF, myocardial infarction, ischemia, decreased level of consciousness, shock, SOB or pulmonary congestion are present).
   A. If ventricular rate is greater than 150 consider immediate cardioversion (rarely needed for rates less than 150).
   B. Consider brief trial of medications based on arrhythmia.
6. If stable treat according to arrhythmia.
   A. Atrial fibrillation/atrial flutter.
      1. Consider digoxin, diltiazem, beta blockers, verapamil, procainamide, quinidine, anticoagulants.
   B. Paroxysmal supraventricular tachycardia (PSVT)
      1. Vagal maneuvers (carotid sinus pressure is contraindicated in patients with carotid bruits; avoid ice water immersion in patients with ischemic heart disease).
      2. Adenosine 6 mg rapid IVP (over 1-3 seconds) may repeat with 12 mg rapid IVP in 1-2 minutes for a total of 30 mg.
      3. Check QRS complex width
         A. If normal complex width
            1. Verapamil 2.5-5.0 mg slow IV, repeat with 5-10 mg in 15-30 minutes if no response.
            2. Consider digoxin, beta blockers, diltiazem.
            3. Consider synchronized cardioversion.
         B. If wide complex width
            1. Lidocaine 1-1.5 mg/kg IVP.
            2. Procainamide 20-30 mg/min, to total of 17 mg/kg.
            3. Consider synchronized cardioversion.
         C. Wide complex tachycardia of uncertain type
            1. Lidocaine 1-1.5 mg/kg IVP, repeat every 5-10 minutes with 0.5-0.75 mg/kg, up to maximum total of 3 mg/kg.
            2. Adenosine 6 mg rapid IVP may repeat with 12 mg rapid IVP in 1-2 minutes for a total of 30 mg.
            3. Procainamide 20-30 mg/min, to total of 17 mg/kg.
            4. Bretylium 5-10 mg/kg IV over 8-10 minutes, maximum total 30 mg/kg over 24 hours.
            5. Consider synchronized cardioversion.
         D. Stable ventricular tachycardia
            1. Lidocaine 1-1.5 mg/kg IVP, repeat every 5-10 minutes with 0.5-0.75 mg/kg, up to maximum total of 3 mg/kg.
            2. Procainamide 20-30 mg/min, to total of 17 mg/kg.
            3. Bretylium 5-10 mg/kg IV over 8-10 minutes, maximum total 30 mg/kg over 24 hours.
            4. Consider synchronized cardioversion.

Electrical Cardioversion Algorithm

1. Consider in tachycardia with serious signs and symptoms related to the tachycardia (seldom needed for ventricular rates less than 150 beats/min).
2. Check oxygen saturation, suction, IV line, airway equipment.
3. Premedicate whenever possible.
4. Synchronized cardioversion (note if conditions are critical, go immediately to unsynchronized cardioversion).
   A. For VT, atrial fib/flutter use 100 J, 200 J, 300 J, 360 J.
   B. For PSVT (and sometimes atrial flutter) start with 50 J.

Ventricular Ectopy

1. Lidocaine 1 mg/kg.
2. If not suppressed, repeat lidocaine 0.5 mg/kg every 2-5 minutes until no ectopy or up to total of 3 mg/kg.
3. If not suppressed, procainamide 20 mg/min until no ectopy or up to total of 1000 mg.
4. If not suppressed, and not contraindicated, bretylium 5-10 mg/kg over 8 minutes.
5. If not suppressed, consider overdrive pacing.

Maintenance Drips

1. After lidocaine dose of 1 mg/kg: start lidocaine drip at 2 mg/min.
2. After lidocaine dose of 1-2 mg/kg: start lidocaine drip at 3 mg/min.
3. After lidocaine dose of 2-3 mg/kg: start lidocaine drip at 4 mg/min.
4. After loading dose of procainamide: start procainamide drip at 1-4 mg/min.
5. After loading dose of bretylium: start bretylium drip at 2 mg/min.

Drugs That May Be Given Endotracheally (mnemonic **ALIEN V**)

A Atropine
L Lidocaine
I Isoproterenol
E Epinephrine
N Naloxone

V Valium

Pediatric ACLS Drugs

Drug	Dose	Remarks
Adenosine	0.1-0.2 mg/kg	Give rapid IV bolus; max single dose 12 mg
Atropine	0.01-0.02 mg/kg Min dose: 0.1 mg	Max single dose: 0.5 mg in child, 1.0 mg in adolescent
Bretylium	5 mg/kg (may be increased to 10 mg/kg)	Give rapid IV Loading dose
Calcium (10%)	20 mg/kg per dose	Give slowly
Dopamine	2-20 mcg/kg/min	Titrate to desired effect
Dobutamine	2-20 mcg/kg/min	Titrate to desired effect
Epinephrine	First dose: IV/IO: 0.01 mg/kg (1:10k) ET: 0.1 mg/kg (1:1000); doses as high as 0.2 mg/kg may be effective Subsequent doses: IV/IO/ET: 0.1 mg/kg	Epinephrine infusion: 0.05-1.0 mcg/kg/min titrate to desired effect
Lidocaine	1 mg/kg per dose	Infusion 20-50 mcg/kg/min
Narcan	0.01 mg/kg	
Sodium Bicarbonate	1-2 mEq/kg per dose or 0.3 x kg x base deficit	Infuse slowly
Valium	0.1-0.25 mg/kg	
Defibrillation	2-4 watt-secs/kg	
Cardioversion	0.25-1.0 watt-secs/kg	

Preoperative Evaluation

1. **Preoperative evaluation**
   - **A.** Preoperative evaluation should include obtaining past medical, surgical, and anesthetic histories, reviewing allergies and current medications, physical exam including airway examination, reviewing of labs, EKG, x-rays and/or other studies, assignment of ASA classification and formulation of the anesthetic plan.
2. **ASA (American Society of Anesthesiology) physical status classification**
   - **A.** The ASA physical status has been shown to generally correlate with the perioperative mortality rate (mortality rates by ASA classification are given below). While not perfect, the ASA physical status remains useful in planning anesthetic management and monitoring techniques.
   - **B. ASA 1:** a normal healthy patient (0.06-0.08%).
   - **C. ASA 2:** a patient with a mild systemic disease (mild diabetes, controlled hypertension, obesity) (0.27-0.4%).
   - **D. ASA 3:** a patient with a severe systemic disease that limits activity (angina, COPD, prior myocardial infarction) (1.8-4.3%).
   - **E. ASA 4:** a patient with an incapacitating disease that is a constant threat to life (CHF, renal failure) (7.8-23%).
   - **F. ASA 5:** a moribund patient not expected to survive 24 hours (ruptured aneurysm) (9.4-51%).
   - **G. ASA 6:** brain-dead patient whose organs are being harvested.
   - **H. For emergent operations**, add the letter E after the classification.
4. **Candidates for preoperative pulmonary function tests (PFT's)**
   - **A.** Patients with any evidence of chronic pulmonary disease.
   - **B.** Heavy smokers with history of persistent cough.
   - **C.** Patients with chest wall and spinal deformities.
   - **D.** Morbidity obese patients.
   - **E.** Elderly patients (>70 years of age).
   - **F.** Patients for thoracic surgery.
5. **Preoperative laboratory evaluations**
   - **A. Hemoglobin:** menstruating females, children less than 1 year old or with suspected sickle cell disease, history of anemia, blood dyscrasia or malignancy, congenital heart disease, chronic disease states, age greater than 60 years.
   - **B. WBC count:** suspected infection or immunosuppression.
   - **C. Platelet count:** history of abnormal bleeding or bruising, liver disease, blood dyscrasias, chemotherapy, hypersplenism.
   - **D. Coagulation studies:** history of abnormal bleeding, anticoagulant drug therapy, liver disease, malabsorption, poor nutritional states.
   - **E. Electrolytes, blood glucose, BUN/creatinine:** patients with hypertension, diabetes, heart disease, or disease states with potential for fluid-electrolyte abnormalities. Patients taking digoxin, diuretics, or ACE-inhibitors. Blood tests performed within 6 months of surgery that show normal results can be used if there has been no intervening clinical event.
   - **F. Liver function tests:** patients with liver disease, history of or

exposure to hepatitis, history of alcohol or drug abuse, drug therapy with agents that may affect liver function.

G. Pregnancy test: patients in whom pregnancy cannot be reliably ruled out by history.

H. Electrocardiogram: men over age 40, women over age 55, history or symptoms of cardiac disease, history of diseases associated with cardiac involvement (hypertension, diabetes, morbid obesity, peripheral vascular disease, collagen vascular disease, cocaine abuse); patients using phenothiazine, antidepressants, doxorubicin; patients at risk for electrolyte abnormalities; patients having elective intrathoracic, intraperitoneal, aortic surgery or emergency operations; patients having major neurosurgery. An EKG showing normal results that was performed within 6 months of surgery can be used if there has been no intervening clinical event.

I. Chest x-ray: patients with symptoms of pulmonary disease, airway obstruction, cardiac disease, malignancy, history of heavy smoking, age greater than 60 years. A study showing normal results that was performed within one year of surgery can be used if there has been no intervening clinical event.

J. Urinalysis: no indication in preanesthetic evaluation; surgeon may request to rule out infection before certain surgical procedures, particularly those involving prosthetic implants.

K. Cervical spine flexion/extension: patients with rheumatoid arthritis or Down's syndrome.

6. Preoperative fasting guidelines (hours)

Age	Clear Liquids	Food/Milk
Prem/Newborn	2	4
1-6 months	2	4
7-36 months	3	6
3-15 years	3	8
Adults	8	8

A. Clear liquids include water, sugar-water, apple juice, non-carbonated soda (not pulp-containing juices, milk, etc).

B. Medications can be taken with a small amount of a clear liquid).

7. Risks of anesthesia

A. Death.

B. Neurologic: stroke.

C. Cardiovascular: perioperative MI has >50% mortality 24-96 hr post-operatively.

D. Pulmonary: aspiration pneumonia (>70% of anesthetic deaths).

E. Renal: usually preexisting.

F. Hepatic: halothane hepatitis [1:40,000 cases].

G. Nonsurgical: damage to nerves, eyes, teeth, mouth, tongue, etc.

H. Exacerbation of existing disease: [eg. multiple sclerosis].

I. Pregnancy: high fetal risk.

8. Cardiovascular disease

A. Preoperative risk factors: the two most important are a history of recent (within 6 months) myocardial infarction and evidence of congestive heart failure.

B. Generally accepted contraindications to elective noncardiac surgery include a myocardial infarction less than 1 month prior to surgery, uncompensated heart failure, and severe aortic or mitral stenosis.

C. Goldman risks factors for a perioperative myocardial infarction

1. Jugular venous distension or S3.
2. Recent myocardial infarction (<6 months).
3. Cardiac rhythm other than normal sinus rhythm.
4. Greater than 5 PVC's per minute.
5. Intraperitoneal, intrathoracic, or aortic surgery.
6. Age greater than 70 years.
7. Significant aortic stenosis.
8. Emergent operation.
9. Poor generalized medical condition.

9. Incidence of perioperative myocardial reinfarction

Time elapsed since prior MI	**Reinfarction Rate (%)**
0-3 months	37
4-6 months	16
greater than 6 months	5

Based on work by Tarhan et al., 1972.
Mortality after perioperative myocardial reinfarction: 20-50%.
Infarction rate in the absence of a prior MI: 0.13%

10. Anesthesia preoperative note

A. Statement of the date and time of the interview, the planned procedure, and a description of any extraordinary circumstances regarding the anesthesia.

B. Relevant past medical/surgical history and laboratory studies.

C. Current medications and allergies.

D. Physical exam, including airway evaluation (see below). Current vitals, height and body weight.

E. Overall impression of the complexity of the patient's medical condition, with assignment to one of the ASA Physical Status Classes (see above).

F. Anesthetic plan (general anesthesia, regional, spinal, etc.). The anesthetic plan is based on the patient's medical status, the planned operation, and patient wishes.

G. Documentation of risks and benefits explained to the patient.

11. Airway evaluation

A. Medical conditions associated with difficult intubations

1. **Arthritis:** patients with arthritis may have a decreased range of neck mobility. Rheumatoid arthritis patients have an increased risk of atlantoaxial subluxation.
2. **Morbid obesity:** obese patients have increased incidence of sleep apnea.
3. **Tumors:** tumors may obstruct the airway or cause extrinsic compression and tracheal deviation.
4. **Infections:** infections of any oral structure may obstruct the airway.
5. **Trauma:** trauma patients are at increased risk for cervical spine injuries, basilar skull fractures, intracranial injuries, and facial bone fractures.
6. **Burns:** increased risk of inhalation injuries.

7. **Down's Syndrome:** Down's patients may have atlantoaxial instability and macroglossia.
8. **Scleroderma:** scleroderma may result in decreased range of motion of the temporomandibular joint and narrowing of the oral aperture.
9. **Acromegaly:** these patients may have overgrowth and enlargement of tongue, epiglottis and vocal cords.
10. **Dwarfism:** associated with increased atlantoaxial instability.
11. **Congenital anomalies:** a variety of congenital syndromes may complicate airway management (such as Pierre Robin syndrome or Treacher Collins syndrome).

B. Physical exam

1. **Anatomic variations:** micrognathia, prognathism, large tongue, arched palate, short neck, prominent upper incisors.
2. **Mouth**
   - **A.** Generally, patients should be able to open their mouth at least 3 finger breadths (50-60 mm).
   - **B.** Poor dentition or loose teeth increase the risk of dental damage and dislodgement.
   - **C.** Macroglossia will increase difficultly of intubation.
3. **Neck**
   - **A. Anterior mandibular space:** estimated by measuring the distance between the hyoid bone and the inside of the mentum or between the notch of the thyroid cartilage to the mentum. An inadequate mandibular space is associated with a hyoid-mentum distance of less than 3 cm or a thyroid notch-mentum distance of less than 6 cm.
   - **B. Cervical spine mobility (atlantooccipital joint extension):** thirty-five degrees of extension are possible at the normal atlantooccipital joint
   - **C**. Presence of a healed or patent tracheostomy stoma.
4. **Airway classification**
   - **A. Mallampati classification**
     1. **Class 1:** able to visualize the soft palate, fauces, uvula, anterior and posterior tonsillar pillars.
     2. **Class 2:** able to visualize the soft palate, fauces, and uvula. The anterior and posterior tonsillar pillars are hidden by the tongue.
     3. **Class 3:** only the soft palate and base of uvula are visible.
     4. **Class 4:** only the soft palate can be seen (the uvula is not visualized).
   - **B. Grades of laryngoscopic view**
     1. **Grade 1:** visualization of the entire laryngeal aperture.
     2. **Grade 2:** visualization of just the posterior portion of the laryngeal aperture.
     3. **Grade 3:** visualization of only the epiglottis.
     4. **Grade 4:** visualization of just the soft palate.

C. Predictors of difficult intubation

1. Obesity.
2. Buckteeth.
3. Large tongue.
4. Decreased jaw movement.
5. Receding mandible or anterior larynx
6. Short stout neck.

Basic Anesthesia

Medical Gas Systems

1. Oxygen

A. Oxygen is stored as a compressed gas at room temperature or refrigerated as a liquid.

B. The pressure in an oxygen cylinder is directly proportional to the volume of oxygen in the cylinder.

2. Nitrous oxide

A. At room temperature, nitrous oxide is stored as a liquid.

B. In contrast to oxygen, the cylinder pressure for nitrous oxide does not indicate the amount of gas remaining in the cylinder. Cylinder pressure remains at 750 psi as long as any liquid nitrous oxide is present (when cylinder pressure begins to fall, only about 400 liters of nitrous oxide remains).

C. The only way to determine residual volume of nitrous oxide is to weigh the cylinder.

3. Air

A. Air is stored as a compressed gas at room temperature.

B. Cylinder air is medical grade and is obtained by blending oxygen and nitrogen.

4. Nitrogen

A. Nitrogen is stored as a compressed gas at room temperature.

Characteristics Of Medical Gas Cylinders

	Oxygen	Nitrous Oxide	Carbon Dioxide	Air	Nitrogen
Cylinder Color	Green	Blue	Gray	Yellow	Black
Form	Gas	Liquid	Liquid	Gas	Gas
Capacity (L)	625	1590	1590	624	625
Pressure (psi)	2000	750	838	1800	2000

Electrical Safety

1. Line isolation monitor

A. Line isolation monitor measures the potential for current flow from the isolated power supply to ground.

B. An alarm is activated if an unacceptably high current flow to ground becomes possible (usually 2 mA or 5 mA).

C. Operating room power supply is isolated from grounds by an isolation transformer. The line source is grounded by the electrical provider while the secondary circuit is intentionally not grounded.

2. Electrical shock

A. Macroshock

1. Macroshock refers to the application of electrical current through intact skin.
2. Currents exceeding 100 mA may result in ventricular fibrillation.
    A. Shock.
    B. Deliberate hypotension (particularly with nitro-prusside).
    C. Atropine (dilates pulmonary vessels and airways).
    D. Anesthesia (nearly all agents except ketamine).
3. Upright posture: increases West's Zone I.
4. Pulmonary embolism.
5. COPD: causes nonvascular air space at the alveolar level.
6. Mechanical obstruction of the pulmonary arteries.
7. Ventilation gas leaving the normal air passages: bronchopleural fistula, tracheal disruption, cuff leak.

B. Microshock

1. Microshock refers to the application of electrical current directly to the heart (as might occur with guide wires or pacing wires).
2. Currents exceeding 50 microamps through a ventricular catheter may induce ventricular fibrillation.
3. The national code requires less than 10 microamps maximum permissible leakage through electrodes or catheters that contact the heart.
4. Line isolation monitors do not protect a patient from microshock.

Anesthesia Machine

1. **Safety valves, regulators, etc**
   - **A. Outlet check value:** prevents gas cylinders from crossfilling.
   - **B. Pressure regulator:** reduces cylinder gas pressure to below 50 psi.
   - **C. Fail-safe valve:** closes nitrous oxide and other gas lines if oxygen pressure falls below 25 psi to prevent accidental delivery of a hypoxic mixture.
   - **D. Diameter index safety system (DISS):** prevents incorrect gas line attachment to the anesthesia machine.
   - **E. Pin index safety system (PISS):** interlink between the anesthesia machine and gas cylinder; prevents incorrect cylinder attachment.
   - **F. Second stage oxygen pressure regulator:** oxygen flow is constant until oxygen pressure drops below 12-16 PSI; whereas other gases shut off if oxygen pressure is less than 30 PSI. This ensures that oxygen is last gas flowing.
2. **Flowmeters**
   - **A.** The flowmeters on anesthesia machines are classified as constant-pressure, variable orifice flowmeters.
3. **Vaporizers**
   - **A. Classification of modern vaporizers**
     1. **Variable bypass:** part of the total gas flow coming into the vaporizer is bypassed into the vaporizing chamber and then returns to join the rest of the gas at the outlet.
     2. **Flow-over:** the gas channeled to the vaporizing chamber flows over the liquid agent and becomes saturated.
     3. **Temperature-compensated:** automatic temperature compensation device helps maintain a constant vaporizer output over a wide range of temperatures.
     4. **Agent specific.**
     5. **Out of circuit:** not in the breathing circuit.
   - **B.** Vaporizer output is not influenced by fresh gas flows until very low flow rates (< 250 ml/min) or very high flow rates (> 15 l/min).
4. **Anesthesia ventilators**
   - **A. Power source:** contemporary ventilators have a pneumatic and electrical power source.
   - **B. Drive mechanism:** compressed gas is the driving mechanism.
   - **C. Cycling mechanism:** time-cycled, and inspiration is triggered by a timingdevice.
   - **D. Bellows classification:** direction of the bellows during expiration determines the classification. Ascending bellows (bellow ascends during expiration) is safer; a ascending bellow will not fill if a disconnect occurs.
   - **E.** Because the ventilator's pressure relief valve is closed during inspiration, the circuit's fresh gas flows contribute to the tidal volume delivered to the patient. The amount each tidal volume will increase: (fresh gas flow ml/min)x(% inspiratory time) divided by the respiratory rate.
   - **F.** The use of the oxygen flush valve during the inspiratory cycle of a ventilator must be avoided because the pressure-relief valve is closed and the surge of circuit pressure will be transferred to the patient's lungs.

Patient Monitors

1. Capnogram

A. The normal end-tidal to arterial CO_2 gradient (dCO_2) is 2-5 mmHg. Reflects alveolar dead space (alveoli ventilated but not perfused).

B. Causes of increased dCO_2

1. Decreased pulmonary arterial pressure: causes a larger West's Zone 1 (region of the lung in which interstitial pressure is greater than pulmonary arteriolar pressure), leading to more unperfused alveoli and more alveolar dead space. Causes include: shock; deliberate hypotension (particularly with nitroprusside); atropine (dilates pulmonary vessels as well as airways); anesthesia (nearly all agents except ketamine).
2. Upright posture: increases West's Zone 1.
3. Pulmonary embolism: air, fat, thrombus, amniotic fluid, etc.
4. COPD: causes nonvascular air space at the alveolar level.
5. Mechanical obstruction of the pulmonary arteries.
6. Ventilation gas leaving the normal air passages: bronchopleural fistula, tracheal disruption, cuff leak.

C. Causes of increased end-tidal CO_2

1. Hypoventilation.
2. Sodium bicarbonate.
3. Laparoscopy (CO_2 inflation).
4. Anesthetic breathing circuit error
   A. Inadequate fresh gas flow.
   B. Rebreathing.
   C. Faulty circle absorber valves.
   D. Exhausted soda lime.
5. Hyperthermia.
6. Improved blood flow to lungs: following resuscitation, after hypotension.
7. Water in capnograph head.

D. Causes of decreased end-tidal CO_2

1. Hyperventilation.
2. Inadequate sampling volume.
3. Incorrect placement of sampling catheter (in fresh gas stream).
4. Hypothermia.
5. Incipient pulmonary edema.
6. Air embolism.
7. Decreased blood flow to lungs.

2. Pulse oximetry

A. Oxyhemoglobin absorbs more infrared light (eg, 660 nm), while deoxyhemoglobin absorbs more red light (eg, 940 nm).

B. The change in light absorption during arterial pulsations is the basis of oximetry determinations. The ratio of the absorptions at a red and infrared wavelength is analyzed by a microprocessor to give the oxygen saturation reading.

C. Oxygen saturation of 90% indicates a PaO_2 of approximately 60 mmHg in normal adults.

D. Because carboxyhemoglobin and oxyhemoglobin absorb light at 660 nm identically, pulse oximeters that only compare two wavelengths of light will register a falsely high reading in patients suffering from carbon monoxide poisoning.

E. Methemoglobin has the same absorption coefficient at both red and infrared wavelengths, resulting in a 1:1 absorption ratio corresponding to a saturation reading of 85%. Thus, methemoglobinemia causes a falsely low saturation reading when SaO_2 is actually greater then 85% and a falsely high reading if SaO_2 is actually less than 85%.

F. Fetal hemoglobin and bilirubin do not affect pulse oximeter.

G. Sources of error

1. **Dyshemoglobins**
   - A. Carboxyhemoglobin: because carboxyhemoglobin and oxyhemoglobin absorb light at 660 nm identically, pulse oximeters that only compare two wavelengths of light will register a falsely high reading in patients suffering from carbon monoxide poisoning.
   - B. Methemoglobin: methemoglobin has the same absorption coefficient at both red and infrared wavelengths, resulting in a 1:1 absorption ratio corresponding to a saturation reading of 85%. Thus, methemoglobinemia causes a falsely low saturation reading when SaO_2 is actually greater then 85% and a falsely high reading if SaO_2 is actually less than 85%.
2. **Intravenous dyes**
   - A. Methylene blue: can cause large, rapid decrease in saturation without decreases in the actual oxygen saturation.
   - B. Indocyanine green causes smaller false decreases in saturation.
   - C. IV fluorescein or indigo carmine have little effect.
3. **Excessive ambient light.** In cases of reduced pulse amplitude, pulse oximeters may become sensitive to external light sources, such as fuorescent room lights.
4. **Motion artifact.**
5. **Venous pulsations.** Pulse oximeter design assumes that the pulsatile component of the light absorbance is due to arterial blood.
6. **Low perfusion.**
7. **Leakage of light** from the light-emitting diode to the photodiode, bypassing the arterial bed.
8. **Penumbra effect.** Pulse oximeters whose sensors are malpositioned may display SaO_2 values in the 90-95 per cent range on normoxemic subjects. This so-called “penumbra effect” can cause underestimation at high saturations, overestimation at low saturations, and a strong dependence of the error on instrument and sensor.

Anesthesia Machine Check

Anesthesia Machine Check List

The anesthesia machine check list, or a reasonable equivalent, should be conducted before administering anesthesia. This is a guideline which users are encouraged to modify to accommodate differences in equipment design and variations in local clinical practice.

1. **Emergency ventilation equipment***
   A. Verify jet ventilator is hooked up and working.
2. **Overview***
   A. Inspect machine for the following
      1. Plugged in.
      2. Flowmeters off.
      3. Vaporizers filled and caps tight.
      4. Tanks on machine properly.
      5. No obvious problems.
      6. Breathing circuit attached.
3. **Electrical systems***
   A. Turn on master switch.
   B. Do battery check.
   C. Turn on all monitors.
4. **High pressure systems***
   A. Check Oxygen cylinders
      1. Disconnect wall pipeline.
      2. Open 0_2 tank.
      3. Open 0_2 flowmeter.
      4. Tank should stay at least 1000 p.s.i..
      5. Close tank.
      6. Low 0_2 pressure alarm should respond as bobbin falls.
      7. Turn off 0_2 flowmeter.
      8. Reconnect pipeline 0_2.
   B. Check pipeline pressures (should read around 50 p.s.i.).
5. **Low pressure systems***
   A. Test flowmeters
      1. Adjust flow of gases through their full range checking for smooth operation.
      2. Check $N_20/0_2$ ratio alarm by trying to create hypoxic mixture (remember to have gas switch on "$N_20/0_2$") and verify correct changes in flow.
   B. Check for low pressure leaks
      1. Drager (no back-check valve)
         A. Turn 0_2 flowmeter to 400 cc/min.
         B. Open vaporizer.
         C. Occlude gas outlet: bobbin should fall.
         D. Open gas outlet: bobbin should rise.
         E. Close vaporizer.
      2. Ohmeda (back-check valve)
         A. Turn of master switch and flowmeters.
         B. Attach suction bulb to common gas outlet.
         C. Squeeze bulb repeatedly until it collapses.
         D. Verify it stays collapsed for 10 seconds.

E. Repeat with vaporizers turned on.
F. Turn on switch and turn off vaporizers.

6. Scavenging system*

A. Adjust and check scavenging system

1. Ensure proper connections between scavenging system and both APL (popoff valve) and ventilator's relief valve.
2. Close scavenge valve, then open 1 1/2 turns.
3. Fully open APL valve and occlude the Y-piece.
4. With minimum 0_2 flow, scavenger reservoir should collapse completely. Verify that PIP valve reads zero (checks negative pressure valve).
5. With 0_2 flush activated, allow the scavenger reservoir bag to distend fully and check that PIP valve reads less than 10 cm/H_20 (checks positive pressure pop-off valve).

7. Breathing system

A. Calibrate 0_2 monitor*

1. Ensure monitor reads 21% room air.
2. Verify low 0_2 alarm is enabled and functioning.
3. Reinstall sensor in circuit and flush breathing system with 100% oxygen.
4. Verify that monitor now reads greater than 90%.

B. Check initial status of system

1. Set selector switch to "bag" mode.
2. Check that breathing circuit is complete, undamaged and unobstructed.
3. Verify $C0_2$ absorbent is adequate.
4. Install any accessory equipment such as humidifier.

C. Perform leak check of breathing circuit

1. Set all gas flows to minimum.
2. Close APL valve and occlude Y piece.
3. Pressurize breathing system to 30 cm H_20 with 0_2 flush.
4. Ensure that pressure remains fixed for at least 10 seconds.
5. Open APL valve and ensure that pressure decreases.

8. Manual and automatic ventilation systems

A. Test ventilation systems and unidirectional valves

1. Set appropriate TV, rate, inspiratory flow.
2. Make sure PEEP valve is off.
3. Set to ventilator mode.
4. Place breathing bag on Y-piece.
5. Turn ventilator on and fill bellows with 0_2 flush valve.
6. Set 0_2 to minimum and other gas flows to “0”.
7. Verify that during inspiration bellow delivers appropriate TV and that bellows empties completely on expiration.
8. Set fresh gas flow to about 5 liters per minute.
9. Verify that the ventilator bellows and simulated lungs fill and empty appropriately without sustained pressure at end expiration.
10. Check for proper action of unidirectional valves.
11. Turn ventilator "off" and switch to manual ventilation.
12. Ventilate manually and assure inflation and deflation of artificial lungs and appropriate feel of system resistance and compliance.

9. Monitors

A. Check, calibrate, and/or set alarm limits of all monitors

1. Capnometer and respiratory volume monitor.
2. 0_2 analyzer.
3. Pulse oximeter.
4. Set-up and calibrate invasive monitor transducers if applicable.
5. Airway pressure high and low alarms.
6. Automatic BP cuff.
7. EKG monitor.
8. Temperature probe available.
9. Transcutaneous 0_2.

10. Final check

A. Check final status of machine

1. Vaporizers off.
2. APL open.
3. Selector switch to "bag".
4. All Flowmeters to zero (0_2 to minimum flow).
5. Patient suction level adequate.
6. Breathing system ready to use.

11. Additional equipment*

A. Additional equipment if needed

1. Blood warmers/Level 1.
2. Bair Hugger.
3. Warming blanket.
4. Operating room table works.
5. Portable oxygen available.

*These steps need not be repeated if same provider uses machine in successive cases

Pharmacology

Basic Pharmacology

1. Stages of general anesthesia

- **A. Stage 1 (amnesia):** Begins with induction of anesthesia and ends with the loss of consciousness (loss of eyelid reflex). Pain perception threshold during this stage is not lowered.
- **B. Stage 2 (delirium/excitement):** Characterized by uninhibited excitation. Agitation, delirium, irregular respiration and breath holding are commonly seen. Pupils are dilated and eyes are divergent. Potentially dangerous responses to noxious stimuli can occur during this stage, including vomiting, laryngospasm, hypertension, tachycardia, and uncontrolled movement.
- **C. Stage 3 (surgical anesthesia):** characterized by central gaze, constricted pupils, and regular respirations. Target depth of anesthesia is sufficient when painful stimulation does not elicit somatic reflexes or deleterious autonomic responses.
- **D. Stage 4 (impending death/overdose)**: onset of apnea, dilated and nonreactive pupils, hypotension to complete failure of the circulation. This stage is commonly referred to as "too deep."

2. Components of general anesthesia

- **A. Unconsciousness (hypnosis).**
- **B. Analgesia (areflexia).**
- **C. Muscle Relaxation.**

3. Pharmacokinetics of inhaled anesthetics

- **A. Concentration:** the fraction of a gas in a mixture is equal to the volume of that gas divided by the total volume of the mixture.
- **B. Partial pressure:** the partial pressure of a component gas in a mixture is equal to the fraction it contributes toward total pressure.
- **C. Minimum alveolar concentration (MAC)**
  - **1.** The minimum alveolar concentration of an inhalation agent necessary to prevent movement in 50% of patients in response to a surgical skin incision.
  - **2.** Minimum alveolar concentrations required to prevent eye opening on verbal command, to prevent movement and coughing in response to endotracheal intubation, and to prevent adrenergic response to skin incision have been defined. These are called MAC Awake, MAC Endotracheal Intubation, and MAC BAR (for blockade of autonomic response). In general, MAC Awake is 50% MAC, MAC Endotracheal Intubation is 130% MAC, and MAC BAR is 150% MAC. MAC Amnesia, 25% MAC, has defined as the concentration that blocks anterograde memory in 50% of awake patients.
  - **3.** MAC values for different volatile agents are additive.
  - **4.** The lower the MAC the more potent the agent.
  - **5.** The highest MACs are found in infants at term to 6 months of age and decrease with both increasing age and prematurity.
  - **6.** Factors that have no effect on MAC: duration of anesthesia, gender, thyroid gland dysfunction, hyperkalemia, hypokalemia, $PaCO_2$ 15-95 mmHg, PaO_2 greater than 38

mmHg, and blood pressure greater than 40 mmHg.

7. Factors that increase MAC: hyperthermia, drugs that increase CNS catecholamines, infants, hypernatremia, and chronic ethanol abuse.
8. Factors that decrease MAC: hypothermia (for every Celsius degree drop in body temperature, MAC decreases approximately 2-5%), preoperative medications, IV anesthetics, neonates, elderly, pregnancy, alpha-2 agonists, acute ethanol ingestion, lithium, cardiopulmonary bypass, neuraxial opioids, and PaO_2 less than 38 mmHg.

D. Alveolar uptake: the rate of alveolar uptake is determined by:

1. **Inspired concentration:** a high inspired anesthetic partial pressure (PI) offsets the impact of uptake and thus accelerates induction of anesthesia. This effect of the high PI is known as the concentration effect.
2. **Alveolar ventilation**: increased ventilation increases the rate of alveolar uptake. The net effect is a more rapid rate of rise in the alveolar partial pressure of an inhaled anesthetic and induction of anesthesia.
3. **Anesthetic breathing system:** the rate of rise of the alveolar partial pressure of an inhaled anesthetic is influenced by (1) the volume of the system, (2) solubility of the inhaled anesthetics into the components of the system, and (3) gas inflow from the anesthetic machine.
4. **Uptake of the inhaled anesthetic**
   - **A. Solubility:** the solubility of inhaled anesthetics is defined as the amount of anesthetic agent required to saturate a unit volume of blood at a given temperature and can be expressed as the blood:gas partition coefficient. The more soluble the agent, the greater the uptake into the pulmonary capillaries. The solubility of the inhalation agent in blood is the most important single factor in determining the speed of induction and recovery in individual patients.
   - **B. Cardiac output:** a high cardiac output results in more rapid uptake such that the rate of rise in the alveolar partial pressure and the speed of induction are slowed.
   - **C. Alveolar to venous partial pressure difference:** a large alveolar to venous gradient enhances the uptake of anesthetic by pulmonary blood and tends to slow the rise in the alveolar partial pressure.

E. Second gas effect: the ability of the large volume uptake of one gas (first gas) to accelerate the rate of rise of the alveolar partial pressure of a concurrently administered companion gas (second gas) is known as the second gas effect.

F. Elimination: most of the inhaled agents are exhaled unchanged by the lungs. Hyperventilation, a small FRC (function residual capacity), a low solubility, a low cardiac output, or a large mixed venous-alveolar tension gradient increases the rate of decay.

G. Diffusion hypoxia: results from dilution of alveolar oxygen concentration by the large amount of nitrous oxide leaving the pulmonary capillary blood at the conclusion of nitrous oxide

administration. This can be prevented by filling the patient's lungs with oxygen at the conclusion of nitrous oxide administration.

4. **Pharmacokinetics of intravenous anesthetics**
   - **A. Volume of distribution**: the apparent volume into which a drug has been distributed is called the volume of distribution. The volume of distribution does not represent a real volume but rather reflects the volume of plasma that would be necessary to account for the observed plasma concentration.
   - **B. Plasma concentration curves**
     1. **Distribution (alpha) phase**: the first phase, or the alpha phase, corresponds to the initial distribution of drug from the circulation to tissues.
     2. **Elimination (beta) phase:** the second phase is characterized by a gradual decline in the plasma concentration of drug and reflects its elimination from the central vascular compartment by renal and hepatic mechanisms.
   - **C. Elimination half-time:** the time necessary for the plasma concentration of drug to decline 50 percent during the elimination phase.
   - **D. Physical characteristics of the drug**
     1. **Highly lipid-soluble drugs** (most intravenous anesthetics) are taken up rapidly by tissues.
     2. **With water soluble agents**, molecular size is an important determinant of diffusibility across plasma membranes.
     3. **Degree of ionization:** the degree of ionization is determined by the pH of the biophase and the pka of the drug. Only nonionized (basic) molecules diffuse across the biological membranes.

Local Anesthetics

1. **Uses of local anesthetics**
   - **A. Topical:** lidocaine, cocaine, tetracaine.
   - **B. Infiltration:** lidocaine, bupivacaine, procaine, chloroprocaine, mepivacaine, etidocaine, prilocaine.
   - **C. Peripheral nerve block:** lidocaine, bupivacaine, procaine, chloroprocaine, mepivacaine, etidocaine, prilocaine.
   - **D. IV regional:** lidocaine, bupivacaine, prilocaine.
   - **E. Epidural:** lidocaine, chloroprocaine, bupivacaine, prilocaine, etidocaine, mepivacaine.
   - **F. Spinal:** lidocaine, tetracaine, procaine, bupivacaine.
2. **Rate of systemic absorption of local anesthetics** (from hi to low)
   - **A**. Intravenous > tracheal > intercostal > caudal > paracervical > epidural > brachial plexus > sciatic/femoral > subcutaneous.
3. **Effects of local anesthetics on organ systems**
   - **A. Cardiac**
     1. Local anesthetics depress myocardial automaticity (spontaneous phase IV depolarization) and reduce the duration of the refractory period.
     2. Cardiac dysrhythmia or circulatory collapse is often presenting sign of local anesthetic overdose during general anesthesia.
     3. Intravascular injection of bupivacaine has produced severe cardiotoxic reactions, including hypotension, atrioventricular

heart block, and dysrhythmias such as ventricular fibrillation. Pregnancy, hypoxemia, and respiratory acidosis are predisposing risk factors. Bupivacaine blocks cardiac sodium channels and alters mitochondrial function: Is high degree of protein binding makes resuscition prolonged and difficult.

B. Respiratory

1. Lidocaine depresses the hypoxic drive (the ventilatory response to low PaO_2).
2. Apnea can result from phrenic and intercostal nerve paralysis or depression of the medullary respiratory center following direct exposure to local anesthetic agents (eg, postretrobulbar apnea syndrome).

C. Cerebral

1. Early symptoms of overdose are circumoral numbness, tongue paresthesia, and dizziness. Sensory complaints may include tinnitus and burred vision. Excitatory signs (eg, restlessness, agitation, nervousness, paranoia) often precede central nervous system depression (slurred speech, drowsiness, and unconsciousness).
2. Tonic-clonic seizures; the excitatory reactions are a result of selective blockade of inhibitory pathways. Respiratory arrest often follows seizure activity.
3. Neurotoxicity
   A. Chloroprocaine has been associated with neurotoxicity. The cause of this neural toxicity may be the low pH of chloroprocaine (pH 3.0) or the old preservative, sodium bisulfate, which has been replaced with disodium ethylenediaminetetraacetate (EDTA).
   B. Repeated doses of 5% lidocaine and 0.5% tetracaine have been associated with neurotoxicity (cauda equina syndrome) following infusion through small-bore catheters used in continuous spinal anesthesia. This may be due to pooling of drug around the cauda equina.

D. Immunologic

1. Allergic or hypersensitivity reactions are very rare with local anesthetics. Esters are more likely to induce an allergic reaction because they are derivatives of p-aminobenzoic acid, a known allergen.
2. Allergic reactions to amides are extremely rare and are probably related to the preservative and not the amide itself. Multidose preparations of amides often contain methylparaben, which has a chemical structure similar to that of p-aminobenzoic acid.

E. Musculoskeletal

1. Local anesthetics are myotoxic when injected directly into skeletal muscle.

D. Neurotoxicity

1. Chloroprocaine used to be formulated with methylparaben (preservative) and sodium bisulfite (antioxidant) as a acidic solution (pH 3.0). This formula of cholorprocaine was found to cause localized nerve damage in several case reports.

The new formula of cholorprocaine contains 0.01% ethylenediaminetetraacetic acid (EDTA) as an antioxidant, and has not been associated with neurotoxicity.

2. 5% lidocaine as been implicating as the cause of cauda-equina synrome when used as a viscous dense solution through a narrow intrathecal catheter. Local anesthetics are packaged at concentrations well above their physiologically effective range, but are commonly diluted for use. If this dilution does not occur, long-term or permanent neural deficits can result.

4. General principles of local anesthetics

A. Mechanism of action of local anesthetics

1. Local anesthetics produce a blockade of nerve impulse by preventing increases in permeability of nerve membranes to sodium ions, slowing the rate of depolarization. Local anesthetics interact directly with specific receptors on the sodium channel, inhibiting sodium ion influx. Local anesthetics do not alter the resting membrane potential or threshold potential.

B. Metabolism

1. Esters

A. Ester local anesthetics are predominantly metabolized by pseudocholinesterase (plasma cholinesterase). Cerebrospinal fluid lacks esterase enzymes, so the termination of action of intrathecally injected ester local anesthetics depends upon their absorption into the bloodstream

B. One metabolite, p-aminobenzoic acid, has been associated with allergic reactions.

2. Amides

A. Amide local anesthetics are metabolized by microsomal enzymes in the liver.

B. Metabolites of prilocaine (o-toluidine derivatives), which accumulate after large doses (greater than 10 mg/kg), convert hemoglobin to methemoglobin. Other drugs that can cause methemoglobin include benzocaine.

B. Physiochemical factors

1. **Lipid solubility:** increased lipid solubility increases potency.
2. **Protein binding:** the greater the protein binding, the longer the duration of action.
3. **pKa:** pKa determines the onset time; the closer the pKa of the local anesthetic is to tissue pH, the greater the fraction of the non-ionized, lipid-soluble form, the faster the onset.

C. Misc concepts

1. **Ion trapping:** ion trapping refers to the accumulation of the ionized form of a local anesthetic in acidic environments due to a pH gradient between the ionized and non-ionized forms. This can occur between a mother and an acidotic fetus (i.e., fetal distress) resulting in the accumulation of local anesthetic in fetal blood.
2. **Minimum concentration of local anesthetic (Cm):** Cm is the minimum concentration of local anesthetic that will block nerve impulse conduction and is analogous to the minimum

alveolar concentration (MAC) of inhalation anesthetics. This measure of relative potency is affected by several factors, including fiber size, type, and myelination; pH (acidic pH antagonizes block); frequency of nerve stimulation (access of local anesthetic to the sodium receptor is enhanced by repeatedly opening the sodium channel): and electrolyte concentration (hypokalemia and hypercalcemia antagonize blockade).

Local Anesthetics: dosages for infiltration anesthesia

		Plain Solution		Epi Solution	
Drug	**Usual Conc %**	**Max Dose (mg)**	**Duration (min)**	**Max Dose (mg)**	**Duration (min)**
Procaine Chloroprocaine	1.0 1.0-2.0	1000 800	30-60 30-45	1000	30-90
Lidocaine Mepivacaine Prilocaine	0.5-2.0 0.5-1.0 0.5-1.0	300 300 500	30-120 45-90 30-90	500 500 600	120-360 120-360 120-360
Bupivacaine Etidocaine	0.25- 0.5 0.5-1.0	175 300	120-240 120-180	225 400	180-420 180-420

Local Anesthetics: dosages for epidural anesthesia

Drug	**Usual Conc %**	**Usual Vol (ml)**	**Total Dose (mg)**	**Onset (min)**	**Duration (min)**
Chloroprocaine	2-3	15-30	300-900	5-15	30-90
Lidocaine	1-2	15-30	150-500	5-15	60-120
Mepivacaine	1-2	15-30	150-300	5-15	60-180
Prilocaine	1-3	15-30	150-600	5-15	60-180
Bupivacaine	0.25- 0.75	25-30	37.5- 225	5-15	120-240
Etidocaine	1.0-1.5	15-30	150-300	5-15	120-240

Local Anesthetics: dosages for spinal anesthesia

Drug	Concen-tration (%)	Dose for T10 Level (mg)	Dose for T6 Level (mg)	Dose for T4 Level (mg)	Duration Plain (min)	Duration w/epi (min)
Procaine	10	75	125	200	30-45	60-75
Lidocaine	5.0	25	50-75	75-100	45-60	60-90
Tetracaine*	0.5**	6-8	8-14	14-20	60-90	120-180
Bupivacaine	0.75	4-6	8-12	12-20	120-150	120-150

*For hypobaric spinal: tetracaine diluted with sterile water up to 0.3% solution.
**Preparation concentration of tetracaine is 1%; tetracaine is diluted with 5.0% glucose for hyperbaric solution and normal saline for isobaric solution

Local Anesthetics: maximum dose

Drug	Max Dose Plain (mg)	Max Dose with Epi (mg)	Max Dose Plain (mg/kg)	Max Dose Epi (mg/kg)
Amides				
Bupivacaine	175		3	
Dibucaine			1	
Etidocaine	300		4	
Lidocaine	300	500	4.5	7
Mepivacaine	300		4.5	7
Prilocaine	400		8	
Esters				
Chloroprocaine	600		12	
Cocaine			3	
Procaine	500		12	
Tetracaine	100		3	

Muscle Relaxants

Drug	ED 95 (mg/kg)	Onset (min)	Duration (min)	Histamine Release	Elimination And Misc
Succinyl-choline	0.25	1	5-10	Rare	Plasma cholinesterase, muscarinic and nicotinic stim
d-Tubo-curarine	0.51	3-5	60-90	+++	70% renal; 20% biliary;autonomic ganglia block
Metocurine	0.28	3-5	60-90	++	80-100% renal; autonomic ganglia blockade
Pan-curonium	0.07	3-5	60-90	None	70% renal; 15-20% liver; muscarinic block
Pipe-curonium	0.07	3-5	60-90	None	
Doxacurium	0.25-0.4	4-6	60-90	None	
Atracurium	0.20	3-5	20-35	+	Hofmann elim & ester hydrolysis, laudanosine
Cis-atracurium	0.05	1-2	60	None	Hofmann elimination
Vecuronium	0.05	3-5	20-35	None	10-20% renal; 40-60% biliary; 20% hepatic
Mivacurium	0.08	2-3	12-20	+	plasma cholinesterase
Gallamine	2.5	4-5	70-80	None	80-100% renal; muscarinic block
Rocuronium	0.3	1-2	20-35	None	10-25% renal; 50-70% biliary; 10-20% hepatic

Muscle Relaxants Continued

		Sup Dose(mg/kg)		Infusions (mcg/kg/min)		
Drug	**Intubate Dose (mg/kg)**	**N_2O/ Opioid**	**Inhal-ation**	**Load Dose (mg/kg)**	**N_2O/ Opioid**	**Inhal-ation**
Succinyl-choline	1.0-1.5			1.0-1.5	60-100	30-50
d-Tubo-curarine	0.6	0.1	0.05			
Metocurine	0.4	0.07	0.04			
Pan-curonium	0.1	0.015	0.007			
Pipe-curonium	0.14					
Doxacurium	0.05-0.08	0.005-0.025				
Atracurium	0.4-0.5	0.1	0.07	0.3-0.5	5-10	3-6
Cis-atracurium	0.15-0.2	0.03			1-2	
Vecuronium	0.08-0.1	0.02	0.015	.06-0.1	1-2	0.6-1.2
Mivacurium	0.15-0.25*	0.1	0.1		5-10	
Rocuronium	0.6-1.2	0.075-0.15			6-10	

*Given in divided doses (0.15 mg/kg followed in 30 seconds by 0.10 mg/kg. For children 2 to 12 years of age the recommended dose of mivacurium is 0.20 mg/kg administered over 5 to 15 seconds.

Neuromuscular Blocking Agents

1. Classifications

A. Depolarizing neuromuscular blocker: succinylcholine mimics the action of acetylcholine by depolarizing the postsynaptic membrane at the neuromuscular junction. Because the postsynaptic receptor is occupied and depolarized, acetylcholine has no effect.

B. Non-depolarizing neuromuscular blocker: these agents act by competitive blockade of the postsynaptic membrane, so that acetylcholine is blocked from the receptors and cannot have a depolarizing effect.

2. Succinylcholine

A. Succinylcholine is the only depolarizing muscle relaxant in general use today and is made up of two joined acetylcholine molecules.

B. Metabolism

1. Rapid onset of action (30-60 seconds) with a short duration of action (5-10 minutes).
2. Succinylcholine is rapidly metabolized by pseudocholinesterase into succinylmonocholine so that only a fraction (approximately 10%) of the injected does ever reaches the neuromuscular junction.
3. As serum levels fall, succinylcholine molecules diffuse away from the neuromuscular junction.

C. Adverse side effects of succinylcholine

1. Cardiac dysrhythmias: sinus bradycardia, junctional rhythm, sinus arrest.
2. Hyperkalemia
   A. Normal muscle releases enough potassium during succinylcholine-induced depolarization to raise serum potassium by 0.5 meq/L.
   B. Massive release of intracellular potassium can result from situations where there is a proliferation of extrajunctional receptors. Extrajunctional receptors are not normally present and are suppressed by normal neural activity. Conditions associated with succinylcholine-induced hyperkalemia include: patients with thermal injuries, massive trauma, severe intra-abdominal infection, neurologic disorders (spinal cord injury, encephalitis, stroke, Guillain-Barre syndrome, severe Parkinson's disease), ruptured cerebral aneurysm, polyneuropathy, myopathies (eg, Duchenne's dystrophy) and tetanus. This potassium release is not reliably prevented by pretreatment with a nondepolarizer.
2. Increased intracranial pressure.
3. Increased intragastric pressure: the increase in intragastic pressure is offset by an increase in lower esophageal sphincter tone.
4. Increased intraocular pressure.
5. Myalgia: most common in healthy female outpatients.
6. Fasciculations: fasciculations can be prevented by pretreatment with a small dose of nondepolarizing relaxant.
7. Myoglobinuria.
8. Trismus.
9. Malignant hyperthermia.
10. Generalized contractions: patients afflicted with myotonia may develop myoclonus after succinylcholine administration.

D. Drug interactions with succinylcholine

1. Cholinesterase inhibitors
   A. By inhibiting acetylcholinesterase, they lead to a higher acetylcholine concentration at the nerve terminal which intensifies depolarization.
   B. By inhibiting pseudocholinesterase, they reduce the hydrolysis of succinylcholine.

C. Echothiophate eye drops and organophosphate pesticides fall into this category.

2. Nondepolarizing muscle relaxants
   A. In general, nondepolarizing muscle relaxants antagonize a depolarizing phase I block. An exception to this interaction is pancuronium, which augments succinylcholine blockade by inhibiting pseudocholinesterase.
3. Other drugs (that potentiate the neuromuscular block)
   A. Antibiotics (streptomycins, colistin, polymyxin, tetracycline, lincomycin, clindamycin), antidysrhythmics (quinidine, lidocaine, calcium channel blockers) antihypertensives (trimethaphan), cholinesterase inhibitors, furosemide (in low doses), inhalational anesthetics, local anesthetics, lithium carbonate, magnesium.

3. Non-depolarizing neuromuscular agents

A. Drugs that potentiate nondepolarizing relaxants

1. All volatile agents.
2. Local anesthetics.
3. Calcium channel blockers.
4. Aminoglycosides.
5. Polymixins.
6. Linosamines.
7. Hexamethonium.
8. Trimethaphan.
9. Immunosuppressants.
10. High-dose benzodiazepines.
11. Dantrolene.
12. Magnesium.

4. Sensitivity to neuromuscular blockade

A. Muscles have different sensitivities to muscle relaxants. The most resistant to most sensitive muscles are: vocal cord; diaphragm;orbicularis oculi; abdominal rectus; adductor pollicis; masseter; pharyngeal; extraocular.

Comparison of Tests of Neuromuscular Function

Test	Estimated Receptors Occupied (%)
Tidal volume	80
Twitch height	75-80
Tetanic stimulation (30 Hz)	75-80
Vital capacity	75-80
Train-of-four	75-80
Tetanic stimulation (100 Hz)	50
Inspiratory force	50
Head lift (5 seconds)	33

Pharmacological Characteristics of Anticholinergic Drugs

	Atropine	Scopolamine	Glycopyrrolate
Tachycardia	+++	+	++
Bronchodilation	++	+	++
Sedation	+	+++	0
Antisialagogue	++	+++	++
Amnesia	+	+++	0

O = No effect; + = Minimal effect; ++ = Moderate effect; +++ = Marked effect

Anticholinergics

1. **Mechanism of action:** anticholinergics competitively block binding by acetylcholine and prevent receptor activation. The cellular effects of acetylcholine, which are mediated through second messengers, are prevented.
2. **Central anticholinergic syndrome**
   - A. Scopolamine and atropine can enter the central nervous system and produce symptoms characterized by restlessness and confusion that may progress to somnolence and unconsciousness. Other systemic manifestations include dry mouth, tachycardia, atropine flush, atropine fever, and impaired vision.
   - B. Physostigmine, a tertiary amine anticholinesterase, is lipid-soluble and effectively reverses central anticholinergic toxicity. An initial dose of 0.01-0.03 mg/kg is recommended and may have to be repeated after 15-30 minutes.
   - C. Glycopyrrolate does not easily cross the blood-brain barrier, and thus is not likely to cause a central anticholinergic syndrome.

Anticholinesterases

	Edrophonium	**Neostigmine**	**Pyridostigmine**	**Physostigmine**
Dose (mg/kg)	0.5-1.0	0.035-0.07 (up to 5 mg)	0.15-0.35	0.01-0.03
Onset (min)	Rapid (1)	Intermed (7)	Delayed (10-13)	
Duration (min)	40-65	55-75	80-130	
Renal Excretion (%)	70	50	75	metabolized by plasma esterases
Atropine (mcg/kg)	7-10	15-30	15-20	usually not needed
Glycopyrrolate (mcg/kg)	do not use	7	7	

Muscarinic side effects of cholinesterase inhibitors

Organ system	**Muscarinic side effect**
Cardiovascular	Decreased heart rate, dysrhythmias
Pulmonary	Bronchospasm, increased bronchial secretions
Cerebral	Diffuse excitation (physostigmine only)
Gastrointestinal	Intestinal spasm, increased salivation
Genitourinary	Increased bladder tone
Ophthalmologic	Pupillary constriction

Benzodiazepines

	Midazolam (Versed)	**Diazepam** (Valium)	**Lorazepam** (Ativan)
Relative Potency	3	1	5
Induction	0.15-0.35 mg/kg	0.3-0.5 mg/kg	0.1 mg/kg
Maintenance	0.05 mg/kg PRN or 0.25-1.5 mcg/kg/min	0.1 mg/kg PRN	0.02 mg/kg PRN
Sedation	0.5-4 mg or 0.07 mg/kg IM; 0.3 mg/kg nasal; 0.5 mg/kg PO	2 mg repeated	0.25 mg repeated
Elimination Half-Time (h)	1-4	21-37	10-20

Benzodiazepines

1. **Mechanism of action**
   - A. Benzodiazepines selectively attach to alpha subunits to enhance the chloride channel gating function of the inhibitory neurotransmitter GABA. Benzodiazepine receptors mostly occur on postsynaptic nerve endings in the central nervous system.
2. **Cardiovascular effects**
   - A. Minimal cardiovascular effects.
   - B. Midazolam tends to reduce blood pressure and peripheral vascular resistance more than diazepam.
3. **Respiratory effects**
   - A. Depression of the ventilatory response to $PaCO_2$.
4. **Cerebral effects**
   - A. Reduced cerebral oxygen consumption, cerebral blood flow and ICP.
   - B. Prevention and control of grand mal seizures.
   - C. Mild muscle relaxation mediated at the spinal cord level.
5. **Misc effects**
   - A. Benzodiazepines reduce MAC (minimum alveolar concentration) by up to 30%.
   - B. Cimetidine reduces metabolism of diazepam.
   - C. Pain during IV/IM injection and thrombophlebitis may occur with diazepam (most likely secondary to its organic solvent propylene glycol).
   - D. Erythromycin inhibits midazolam metabolism.
   - E. Heparin displaces diazepam from protein binding sites and increases the free drug concentration.

6. Reversal

A. Benzodiazepines can be reversed by flumazenil (Romazicon).
B. Flumazenil is an competitive inhibitor of GABA.
C. For reversal of conscious sedation: 0.2 mg IV over 15 seconds. Give additional 0.1 mg IV bolus every 60 seconds to achieve desired effect, to a total of 1 mg. For reversal of overdose: 0.2 mg IV over 30 seconds. If necessary, further 0.3 mg IV 60 seconds later. If no effect, 0.5 mg boluses every 60 seconds to a total of 3 mg. For reversal of resedation: 0.2 mg IV as required, to a total of 1 mg/hr, or infusion 0.5 mg/hr.
D. Duration of antagonism is brief and may require repeated doses of reversal.
E. May induce seizures, acute withdrawal, nausea, dizziness, agitation, arrhythmias (particularly in the presence of tricyclic antidepressants).

Opioids

	Meperidine	**Morphine**	**Fentanyl**	**Sufenta**	**Alfentanil**
Equivalent Potency	0.1	1	75 to 125	500-1000	25
Rapid Distribution Half-Time (min)		1.2-2.5	1.4-1.7	1.4	1-3.5
Slow Distribution Half-Time (min)		9-13.3	13-28	17.7	8.2-16.8
Elimination Half-Time (min)	180-264	102-132	185-219	148-164	70-98
Clearance (ml/min/kg)	10-17	14.7	11.6	12.7	6.4
Vol of Distribution (l/kg)	2.8-4.2	3.2	4.1	2.86	0.86
Partition Coefficient (lipid solubility)			816	1727	129
Protein Binding (%)		26-36	79-87	92.5	89-92

Opioids

1. Classification of opioid receptors

- **A. Mu receptor:** morphine is the prototype exogenous ligand.
  - **1. Mu-1:** the main action at this receptor is analgesia, but also responsible for miosis, nausea/vomiting, urinary retention, and pruritus. The endogenous ligands are enkephalins.
  - **2. Mu-2:** respiratory depression, euphoria, sedation, bradycardia, ileus and physical dependence are elicited by binding at this receptor.
- **B. Delta:** modulation of mu receptor, physical dependence. High selective for the endogenous enkephalins, but opioid drugs still bind (leu-enkephalin and beta-endorphin).
- **C. Kappa:** ketocyclazocine and dynorphin are the prototype exogenous and endogenous ligands, respectively. Analgesia, sedation, dysphoria, and psychomimetic effects are produced by this receptor. Binding to the kappa receptor inhibits release of vasopressin and thus promotes diuresis. Pure kappa agonists do not produce respiratory depression.
- **D. Sigma:** N-allylnormetazocine is the prototype exogenous ligand. While this receptor binds many types of compounds, only levorotatory opioid isomers have opioid activity. The sigma receptor binds primarily dextrorotatory compounds. Dysphoria, hypertonia, tachycardia, tachypnea, and mydriasis are the principal effects of this receptor.

2. Opioid systemic effects

- **A. CNS:** CNS depression; pupillary constriction; nausea/vomiting (stimulates chemoreceptor trigger zone); hyperactive spinal reflexes; CNS excitation and seizures (high dose meperidine); analgesia; depressed cough reflex.
- **B. Cardiac:** bradycardia (stimulation of vagal nucleus in medulla); tachycardia (meperidine); arteriolar and venous dilation (orthostatic hypotension); histamine release (morphine and meperidine); cardiac depression (meperidine).
- **C. Respiratory:** increased arterial carbon dioxide tension; decreased breathing rate; increased tidal volume; decreased minute ventilation; decreased ventilatory response to carbon dioxide (displacement of the CO_2 response curve to the right); chest wall rigidity.
- **D. Gastrointestinal:** urinary retention; slow gastric emptying, spasm of sphincter of Oddi (less with meperidine).
- **E. Endocrine:** may block stress response to surgery at high doses.
- **F. Skeletal Muscle:** high doses may cause spasm of the thoracoabdominal muscles (chest wall rigidity).
- **G. Genitourinary tract:** increases tone of ureter and vesicle sphincter, making voiding difficult (can be reversed with atropine).
- **H. Placenta:** opioids can cross the placenta, causing possible neonatal depression.

Alfenta Dosing

	Incremental	Incremental	Continuous	Induction*
Duration	<30 min	30-60 min	>45 min	>45 min
Initial Dose	8-20 mcg/kg	20-50 mcg/kg	50-75 mcg/kg	130-245 mcg/kg
Main-tenance	3-5 mcg/kg or 0.5-1 mcg/kg/min	5-15 mcg/kg	0.5-3.0 mcg/kg/min	0.5-1 mcg/kg/min or general anesthesia
Total Dose	8-40 mcg/kg	up to 75 mcg/kg	depends on time	depends on time
Ventilation	spent or assisted	assist or control	assist or control	assist or control

*Following an anesthetic induction dose of alfentanil requirements for volatile inhalation anesthetics or alfentanil infusion are reduced by 30-50% for the first hour of maintenance.

**Dilution instructions: # ml of Alfenta = 0.1 x pts wt (kg), add this with IV fluids to a final volume of 10 cc for final concentration of 5 mcg/kg/ml.

***Turn off infusion approximately 45 minutes prior to anticipated extubation.

Narcotic/Opioid Techniques

1. Alfenta/Propofol technique (for generally healthy patient)

Induction: 50 mcg/kg Alfenta, and 2 mg/kg propofol.

Muscle relaxant: 0.1 mg/kg vecuronium or relaxant of choice.

Maintenance anesthesia: Alfenta: 25-150 mcg/kg/hr or 0.5-3.0 mcg/kg/min; propofol 6-12 mg/kg/hr or 0.1-0.2 mg/kg/min (generally start at 10 or 12 mg/kg/hr and titrate down to effect).

Dosing plan: turn off Alfenta approximately 30-45 minutes before extubation and turnoff propofol approximately 10-20 minutes before anticipated extubation.

2. Fentanyl dosing

Induction: 2-8 mcg/kg fentanyl combined with thiopental (1-4 mg/kg) and muscle relaxant.

For narcotic induction (balanced technique): surgery time (minutes) divided by 10 equals mcg/kg load (with nitrous, O_2, relaxant).

Maintenance anesthesia: intermittent bolus (25-50 mcg every 15-60 minutes) or continuous infusion (0.5-4.0 mcg/kg/hr) combined with nitrous 70%/oxygen 30%.

Dosing plan: turn off infusion 30 minutes prior to end of case.

3. Sufentanil dosing

A. Analgesic doses for surgical procedures lasting 1-8 hours

Total dosage: 1-8 mcg/kg based on the expected duration of surgery (1-8 hours). Total dosage requirements based on 1 mcg/kg/hr or less.

Maintenance anesthesia: intermittent bolus 10-50 mcg or continuous infusion 0.12-1.0 mcg/kg/hr.

Dilution for infusion: mix 5 cc of Sufenta with 245 cc of crystalloid = concentration of 1 mcg/cc.

Dosing plan: give approximately 75% of total dose as initial dose and combine with nitrous oxide, oxygen, and muscle relaxant; stop infusion 30 minutes prior to extubation.

B. Anesthetic doses for cardiovascular procedures

Total dosage: 8-30 mcg/kg (usually 5-10 mcg/kg during induction), may be given in divided doses prior to intubation, incision, sternotomy, bypass, and upon rewarming. May also be given by an infusion following an initial loading dose.

Maintenance anesthesia: 25-50 mcg as needed.

Dosing plan: administered with oxygen and muscle relaxant.

C. Alternate dosing for sufentanil

Induction: 0.25-2.0 mcg/kg Sufenta (1.0-1.5 mcg/kg) with thiopental (1-4 mg/kg).

For narcotic induction (balanced technique): surgery time (minutes) divided by 100 equals mcg/kg load (with nitrous, O_2, relaxant).

Maintenance anesthesia: intermittent bolus (0.1-0.25 mcg/kg) or continuous infusion (0.25-1.5 mcg/kg/hr, start at 0.3 mcg/kg/hr) combined with nitrous 70%/oxygen 30%, and muscle relaxant.

Opioid Antagonist

1. Naloxone and naltrexone

A. Pure opioid antagonists with a high affinity for mu receptors.

B. Administration results in displacement of opioid agonists from opioid receptors.

C. In contrast to naloxone, naltrexone is effective orally, producing sustained antagonism of the effects of opioid agonists up to 24 hours.

2. Naloxone

A. 1-4 mcg/kg IV will reverse opioid-induced analgesia and respiratory depression.

B. Continuous infusion, 5 mcg/kg/hr IV, will prevent respiratory depression without altering the analgesia produced by neuraxial opioids.

C. Side effects: sudden antagonism can activate the sympathetic nervous system, resulting in cardiovascular stimulation.

Remifentanil (Ultiva)

1. Dosing

A. General anesthesia: an initial dose of 0.5-1 mcg/kg may be administered over 30-60 seconds if endotracheal intubation is to occur less than 8 minutes after the start of the infusion of remifentanil. Continuous infusion dose range 0.05-2 mcg/kg/min depending on which other agents are combined with remifentanil for maintenance anesthesia.

B. Monitored anesthesia care (MAC): remifentanil may be administered as a single dose of 0.5-1.0 mcg/kg (over 30-60 seconds) 90 seconds prior to the placement of a local or regional anesthetic block.

2. Special considerations

A. Muscle rigidity: muscle rigidity cab by caused by remifentanil and is related to the dose and speed of administration. The occurance of muscle rigidity is markedly reduced by administering hypnotics and/or neuromuscular blocking agent prior to or in conjuction with remifentanil.

B. Hypotension and bradycardia: both have been reported and respond to decreases in the administration of remifentanil, IV fluids or catecholamine administration.

C. Rapid offset of action: within 5-10 minutes after the iscontinuation of remifentanil, no residual opioid activity will be present.

3. Special patient populations

A. Pediatric patients: no differences in pharmacokinetics were noted in children. The safety of remifentanil in children under the age of 2 years has not been established.

B. Patients with renal or hepatic impairment: no clinical differences were noted compared to healthy patients.

C. Obese patients: dosing should be based on ideal, rather than actual, body weight in obese patients.

D. Elderly patients: elderly patients are more sensitive to the effects of opioids; therefore, a 50% reduction in dose is recommended for elderly patients.

E. Patients with cholinesterase deficiency: remifentanil is not metabolised by plasma cholinesterase and is not appreciably metabolized by the liver or lung. Remifentanil is expected to have a normal duration of action in patients with atypical cholinesterase.

Intravenous Induction Agents

1. Sodium thiopental (Pentothal) and other barbiturates

A. Preparation: a barbiturate; prepared as a 2.5% solution; water-soluble; pH of 10.5; stable for up to 1-2 weeks if refrigerated.

B. Mechanism of action: depress the reticular activating system, reflecting the ability of barbiturates to decrease the rate of dissociation of the inhibitory neurotransmitter gamma-aminobutyric acid from its receptors.

C. Pharmacokinetics

1. Prompt awakening following IV bolus reflects redistribution from the brain to inactive tissues sites. Ultimate elimination depends almost entirely on metabolism, as less than 1% is recovered unchanged in the urine.
2. Protein binding parallels lipid solubility, decreased protein binding increases drug sensitivity.
3. Protein binding of thiopental in neonates is about half that in adults, suggesting a possible increased sensitivity to this drug in neonates.
4. Fat is the only compartment in which thiopental continues to accumulate 30 minutes after injection.

D. Effects on organ systems

1. Cardiovascular: induction doses cause a decrease in blood pressure and an elevation in heart rate. Tachycardia is probably due to a central vagolytic effect. The cardiac effects of barbiturates can vary markedly depending on the

volume status, baseline autonomic tone, and preexisting cardiovascular disease.

2. **Respiratory:** barbiturate depression on the medullary ventilatory center decreases the ventilatory response to hypercapnia and hypoxia. Laryngospasm and hiccuping are more common after methohexital than after thiopental. Barbiturates do not completely depress noxious airway reflexes.
3. **Cerebral:** barbiturates constrict cerebral vasculature, decreasing cerebral blood flow and intracranial pressure. Barbiturates cause a decline in cerebral oxygen consumption (up to 50% of normal) and slowing of the EEG (progresses from low-voltage fast activity with small doses to high-voltage slow activity and electrical silence with very large doses). This effect may provide some brain protection from transient episodes of focal ischemia (eg, cerebral embolism) but probably not from global ischemia (eg, cardiac arrest). Barbiturates have an antianalgesic effect by lowering the pain threshold.
4. **Renal:** barbiturates decrease renal blood flow and glomerular filtration rate in proportion to the fall in blood pressure.
5. **Hepatic:** hepatic blood flow is decreased. The induction of aminolevulinic acid synthetase stimulates the formation of porphyrin (an intermediary in heme synthesis), which may precipitate acute intermittent porphyria or variegate porphyria in susceptible individuals.

2. Ketamine

A. Mechanism of action: ketamine blocks polysynaptic reflexes in the spinal cord and inhibiting excitatory neurotransmitter effects. Ketamine functionally dissociates the thalamus from the limbic cortex (some brain neurons are inhibited, while others are tonically excited. This produces a state of dissociative anesthesia.

B. Structure: ketamine is a structural analogue of phencyclidine (PCP).

C. Pharmacokinetics: peak plasma levels are achieved within 10-15 minutes after intermuscular injection. Awakening is due to redistribution to peripheral compartments.

D. Effects on organ systems

1. **Cardiovascular:** ketamine increases arterial blood pressure, heart rate, and cardiac output. These indirect cardiovascular effects are due to central stimulation of the sympathetic nervous system. Ketamines direct myocardial depressant effects (large doses) are unmasked by sympathetic blockade or patients who are catecholamine depleted.
2. **Respiratory:** ventilation is minimally affected with normal doses of ketamine. Ketamine is a potent bronchodilator. Increased salivation is associated with ketamine and can be attenuated by pretreating with an anticholinergic.
3. **Cerebral:** ketamine increases cerebral oxygen consumption, cerebral blood flow, and intracranial pressure.

E. Drug interactions: nondepolarizing muscle relaxants are potentiated by ketamine. The combination of ketamine and theophylline may predispose patients to seizures.

3. **Etomidate**
   - **A. Mechanisms of action:** etomidate depresses the reticular activating system and mimics the inhibitory effects of gamma-aminobutyric acid. Etomidates disinhibitory effects on the parts of the nervous system that controls extrapyramidal motor activity contribute to the high incidence of myoclonus.
   - **B. Pharmacokinetics:** like other barbiturates, redistribution is responsible for decreasing the plasma concentration to awakening levels. Biotransformation is five times greater for etomidate than for thiopental.
   - **C. Effects on organ systems**
     1. **Cardiovascular:** minimal cardiovascular changes are seen. Etomidate does not release histamine.
     2. **Respiratory:** ventilation is less affected with etomidate than thiopental.
     3. **Cerebral:** decreases the cerebral metabolic rate, cerebral blood flow, and intracranial pressure. Cerebral perfusion pressure is well maintained. High incidence of nausea/vomiting for etomidate induction.
     4. **Endocrine:** induction doses of etomidate transiently inhibit enzymes involved in cortisol and aldosterone synthesis. Long term infusions lead to adrenocortical suppression.
   - **D. Drug interactions:** fentanyl increases the plasma level and prolongs the elimination half-life of etomidate. Opioids decrease the incidence of myoclonus.
4. **Propofol**
   - **A. Mechanisms of action:** propofol increases the inhibitory neurotransmission mediated by gamma-aminobutyric acid.
   - **B. Structure:** propofol is not water-soluble.
   - **C. Pharmacokinetics:** highly lipid solubility. Rapid awakening results from a very short initial distribution half-life (2-8 minutes). Recovery from propofol is more rapid and accompanied by less hangover than other induction agents.
   - **D. Effects on organ systems**
     1. **Cardiovascular:** decrease in arterial blood pressure owing to a drop in systemic vascular resistance, contractility, and preload. Hypotension is more pronounced than with thiopental. Propofol markedly impairs the normal arterial baroreflex response to hypotension.
     2. **Respiratory:** propofol results in profound respiratory depression. Propofol induced depression of upper airway reflexes exceeds that of thiopental.
     3. **Cerebral:** decreases cerebral blood flow and intracranial pressure. Propofol has antiemetic and antipruritic properties, but does not have anticonvulsant properties.

Inhaled Anesthetics

Agent	MAC[1]	Blood:gas coefficient[2]	Vapor pressure[3]	Metabolism (%)[4]
Methoxyflurane	0.16	12.0	22.5	50
Isoflurane	1.15	1.4	240	0.2
Enflurane	1.68	1.9	172	2-5
Halothane	0.75	2.4	244	15-20
Desflurane	7.25	0.42	669	<0.1
Sevoflurane	2.05	0.68	160	
Nitrous Oxide	105-110	0.46		0.0004

[1]MAC: Minimum alveolar concentration at one atmosphere at which 50% of patients do not move in response to a surgical skin incision; these MAC values are for 30-55 year old human subjects are expressed as a percentage of 1 atm; high altitude requires a higher inspired concentration of anesthetic to achieve the same partial pressure.
[2]Blood:gas partition coefficient is inversely related to the rate of induction.
[3]Vapor pressure is reported as mmHg at 20°C.
[4]Metabolism equals percentage of absorbed anesthetic undergoing metabolism.

Circulatory Effects of Inhaled Anesthetics

	Isoflurane/ Desflurane	Halothane	Enflurane	Nitrous Oxide
Cardiac Output	0	-*	-*	+
Heart Rate	++/0	0	++*	*
Blood Pressure	--*	-*	--*	+
Stroke Volume	-*	-*	--*	-
Contractility	--*	---*	--*	-*
System Vascular Resistance	--	0	-	0
Pulmonary Vascular Resistance	0	0	0	+
Coronary Blood Flow	+	0	0	0
Cerebral Blood Flow	+	+++	+	0
Catecholamine Levels	0	0	0	0

*=Dose dependent; -=decrease; --=large decrease; 0=no change; +=increase; ++=large increase.

Inhaled Anesthetics

A. Ventilation effects of inhalational anesthetics

1. **Breathing patterns:** increased respiratory rate (due to CNS stimulation); decreased tidal volume; decreased minute ventilation (increased respiratory rate insufficient to compensate for decrease TV); overall breathing pattern: rapid, shallow, rhythmic, and regular.
2. **$PaCO_2$:** dose dependent increase except for nitrous (enflurane > halothane or isoflurane); lessens with time.
3. **Ventilatory responsiveness to $PaCO_2$**: threshold increased, decrease slope, decrease sensitivity to $PaCO_2$ (nitrous has least effect).
4. **Ventilatory response to decreasing PaO_2:** inhaled anesthetics inhibit hypoxic drive of carotid body.
5. **Bronchodilation:** probably due to CNS depression or blocking of afferent nerves.

B. Circulatory effects of inhalational anesthetics

1. **Blood pressure**
   A. A greater decrease occurs with isoflurane and enflurane than with halothane; no change with nitrous. Desflurane and sevoflurane also decrease blood pressure in dose-dependent fashion
   B. Halothane and enflurane: decrease contractility and CO.
   C. Isoflurane: decrease SVR and vasodilatation.
2. **Heart rate**
   A. Halothane: no change (baroreceptors inhibited).
   B. Isoflurane: increase by as much as 20% (dose independent increase effect above 1 MAC).
   C. Enflurane: only anesthetic with dose dependent increase (reflex tachycardia via baroreceptor).
   D. Nitrous Oxide: minimally increased.
   E. Desflurane and sevoflurane: increased
3. **Cardiac output:** halothane, but not the other inhalational anesthetics, decrease cardiac output in a dose dependent fashion.decrease. No change to slight increase with nitous oxide.
4. **Stroke volume**: all inhaled anesthetics have a dose dependent reduction (greatest with enflurane), except nitrous (no change).
5. **Contractility:** decrease with halothane and enflurane (min change with isoflurane).
6. **Systemic vascular resistance:** decrease with isoflurane, desflurane, sevoflurane and enflurane (minimal); no change with nitrous or halothane.
7. **Pulmonary vascular resistance:** nitrous increases PVR (especially in patients with pulmonary HTN); halothane, enflurane, and isoflurane have minimal effect.
8. **Right atrial pressure:** increase with nitrous (due to increase in PVR), enflurane and halothane; minimal to no change with isoflurane due to its peripheral vasodilating effects.
9. **Coronary resistance:** decrease with isoflurane (coronary steal syndrome), no significant change with halothane or enflurane, autoregulation remains intact.
10. **Cardiac dysrhythmias**: most with halothane and least with enflurane; halothane, enflurane, and isoflurane all slow conduction through the A-V node; halothane slows conduction through His-Purkinje fibers and is not dose related (arrhythmias should not be treated by decreasing halothane); Prazosin (alpha-1 receptor blocker) increases concentration of epinephrine needed to induce premature ventricular contractions.

C. Metabolic effects of inhalational anesthetics

1. **Halothane:** primarily metabolism (mainly oxidative) and ventilation; oxidative metabolism results in trifluroacetic acid and bromide (Br concentration increases 0.5 mEq/L/MAC hr) with CNS toxicity (somnolence, confusion) occurring with levels greater then 6.0 mEq/L; reductive metabolism (occurs with hypoxia or induction of microsomal enzymes) results in reactive intermediates and fluoride.
2. **Enflurane:** primarily ventilation; minimal metabolism (reduction); oxidative metabolism results in fluoride; enzyme induction with

phenobarbital does not increase defluoridation; defluoridation is increased in patients on chronic INH therapy.

3. **Isoflurane:** primarily ventilation; oxidative metabolism results in trifluroacetic acid and difluromethanol (which is unstable, leading to formic acid and fluoride); enzyme induction does not significantly increase metabolism.
4. **Methoxyflurane:** primarily metabolism (oxidation); major metabolite is fluoride; serum fluoride concentration is lower in pediatric patients due to bone uptake.
5. **Nitrous oxide**: primarily ventilation; minimal reductive metabolism in GI tract by anaerobic bacteria (Pseudomonas).
6. **Desflurane**: primarily ventilation; no evidence of any nephrotoxic effects.
7. **Metabolism** is dependent on the cytochrome P-450 enzyme which is located in the hepatic endoplasmic reticulum and can by induced by drugs (inhaled anesthetics, phenobarbital) and chemicals (polychlorobiphenyls).
8. **Cirrhosis and CHF** decrease metabolism by decreasing hepatic blood flow; cirrhosis also may result in decreased enzyme activity due to parenchymal damage.
9. **Obesity** is associated with increased defluoridation.

D. Halothane hepatic dysfunction

1. **Two entities**
   A. Mild/transient form unrelated to the anesthetic but rather to hypoxia.
   B. Fulminant form possibly secondary to allergic reaction.
2. Most often occurs in middle aged obese females with recent prior exposure to halothane (up to 4 months) resulting in post-operative fever and elevated liver function tests.
3. **Pediatric patients** are less likely to have halothane related hepatic dysfunction even with repeated exposure at short intervals.

E. Fluoride induced nephrotoxicity

1. Seen with fluoride levels greater then 50 uM/L (toxic levels of fluoride levels may be seen after 2.5 MAC hrs of methoxyflurane or 9.6 MAC hrs of enflurane); fluoride nephrotoxicity depends on both the duration of exposure of renal tubules to fluoride as well as the absolute level.
2. Fluoride elimination is dependent on GFR.
3. Fluoride nephrotoxicity may result in inability to concentrate urine.
4. Proposed mechanisms: fluoride induced inhibition of adenylate cyclase in distal convoluted tubules (necessary for ADH action) or possibly fluoride induced intrarenal vasodilation; results in increased medullary blood flow interfering with countercurrent mechanism.

F. Inhaled anesthetics: miscellaneous information

1. **Nitrous oxide:** colorless; odorless; good analgesic.
2. **Halothane:**colorless, poor analgesic; bronchodilation; hypotension (secondary to peripheral vasodilation, myocardial depression; sympathetic ganglionic blockade; and inhibition of the baroreceptor reflex); can cause uterine relaxation; preferentially depresses intercostal muscles more than diaphragm.
3. **Enflurane**: pleasant odor; slightly irritating to upper airways; 2%

patients exhibit EEG patterns of seizure activity.

4. **Isoflurane:** least blood soluble; least potent cardiac depressant; good for controlled hypotension; least increase in cerebral blood flow.
5. **Desflurane:** similar in structure to isoflurane; boils at room temperature at high altitudes (high vapor pressure); the ultrashort duration of action and moderate potency are most characteristic features.

Common Premedications

Classification	Drug	Adult (mg)	Peds (mg/kg)	Route
Barbiturates	Pentobarbital	50-150	2	IM
	Secobarbital		2	IM
	Methohexital		8	IM
	Methohexital 1%			15PR
	Methohexital 10%		25	PR
Non-Barb	Ketamine		2-4	IM
	Ketamine		3	Intranasally
	Ketamine		6-10	PO
Opioids	Fentanyl		0.01-0.02	Oral
	EMLA		2.5 gms	Transdermal
	Sufentanil		0.0015-.0003	Intranasally
	Morphine	5-15	0.05-0.2	IM
	Meperidine	50-100	1-1.5	IM
Benzo-diazipines	Diazepam	5-10	0.2-0.4	PO
	Midazolam	2.5-5		IM
	Midazolam		0.2-0.5	Intranasally
	Midazolam		0.4-1.0	PO
	Midazolam	0.2		Sublingual
	Midazolam		0.5-1.0	rectal
	Lorazepam	2-4		PO, IV
Non-Benzo	Chloral Hydrate	75		PO
Antihistamines	Benadryl	25-75		PO, IM
	Phenergan	25-50	0.5	IM
	Vistaril	50-100	0.5-1.0	IM
Anticholinergic	Atropine	0.3-0.6	0.02	IM, IV
	Scopolamine	0.3-0.6	0.008	IM, IV
	Glycopyrrolate	0.2-0.3	0.004-0.008	IM, IV
H_2 Blockers	Tagamet	300		PO, IM, IV
	Zantac	150		PO
		50		IM, IV
Antacids	Bicitra	15-30 cc		PO
Gastric Stim	Reglan	10-20		PO, IV, IM

Premedications

1. Routine premedications: healthy adults generally do not require premedications prior to going to the operating room or arriving in the preoperative holding area. Occasionally a PO anxiolytic may given. Patients for cardiac surgery, however, typically receive premedications (see cardiac surgery section).

Selected Drugs/Drips

Adenosine (Adenocard)

Actions:an endogenous nucleoside with antiarrhythmic activity, adenosine slows conduction through the A-V node.

Indications: paroxysmal supraventricular tachycardia, Wolff-Parkinson-White syndrome

Dose: 6 mg rapid IV bolus; may be repeated within 1-2 minutes with 12 mg (up to two doses). Children 0.05-0.25 mg/kg.

Complications: the effects are antagonized by methylxanthines (i.e., theophylline).

Misc: contraindicated in patients with second or third-degree heart block or sick sinus syndrome. Large doses given by infusion may cause hypotension. Not effective in atrial flutter or fibrillation

Aminocaproic Acid (Amicar)

Actions: inhibits plasminogen activators (fibrinolysis inhibitor).

Indications: excessive acute bleeding from hyperfibrinolysis, chronic bleeding tendency; antidote for excessive thrombolysis due to streptokinase or urokinase.

Dose: loading dose of 100-150 mg/kg IV over the first 30-60 minutes followed by constant infusion of 1 gm/hour for about 8 hours or until bleeding controlled. Most common regimen for the average adult patient: 5 gram loading (started prior to skin incision) followed by constant infusion of 1 gm/hour.

Complications: contraindicated in patients with active intravascular clotting, DIC, and bleeding in the kidneys or ureters; hypotension, bradycardia, dysrhythmias, elevated LFT's, thrombosis.

Aminophylline

Actions: inhibition of phosphodiesterase, resulting in bronchodilation with positive inotropic and chronotropic effects.

Loading dose: 5-7 mg/kg IVPB over 15-30 min (6 mg/kg PO).

Standard conc: 250 mg/250 D_5W = 1 mg/cc.

Maintenance (IV): 1) Children 1-9 years 1 mg/kg/hr
2) Children >9 years 0.8 mg/kg/hr
3) Adult smokers 0.8 mg/kg/hr
4) Adult non-smokers 0.5 mg/kg/hr
5) Adults w/CHF/liver 0.25 mg/kg/hr

Rate determination: cc/hr = dose x body wt (kg) when mixed 1 mg/kg.

Therapeutic level: 10-20 mg/dl.

Complications: nausea, vomiting, anorexia, dizziness, headaches, agitation, tachyarrhythmias, ventricular arrhythmias, palpitations, overdosage hyperreflexia, convulsions, hypotension, tachypnea.

Misc: aminophylline contains about 80% theophylline by weight.

Amrinone (Inocor)

Actions: phosphodiesterase inhibitor (rapid inotropic agent) causing increase in cardiac output while pulmonary vascular resistance and preload decrease (positive inotropic and vasodilator properties).

Indications: severe CHF not responding to conventional therapy.

Dosage: loading dose of 0.75 mg/kg is given over 3-5 minutes, followed by a 2-20 mcg/kg/min infusion.

Standard conc: 100 mg in 250 cc NS (do not mix in dextrose solutions).
Complications: worsening myocardial ischemia, thrombocytopenia; contraindicated if allergic to bisulfites; hepatic function abnormalities, nausea, vomiting, hypotension, arrhythmias.
Misc.: do not dilute in dextrose containing solutions; do not administer furosemide (lasix) in same IV line.

Aprotinin (Trasylol)
Actions: inhibitor of several proteases (including trypsin, kallikrein, and plasmin) and inhibits factor XIIa activation of complement.
Indications: prophylactic use to reduce bleeding and transfusion requirements in high-risk cardiac surgery patients (i.e., redo CABG and/or valve replacements). Selected use in primary CABG patients is based on the risk bleeding (i.e., patients with impaired hemostasis) or where transfusion is unavailable or unacceptable.
Test dose: 1 ml (1.4 mg) administer IV at least 10 minutes before loading dose. After the test dose is given, either the low dose regimen (100 cc load followed by 25 cc/hr maintenance) or high dose (200 cc load followed by 50 cc/hr maintenance) regimen may be started.
Loading dose: 100 cc (140 mg) or 200 ml (280 mg) IV, slowly over 20-30 minutes with patient in supine position.
Maintenance: 25 (35 mg/hr) or 50 ml/hr (70 mg/hr) started after loading dose is completed.
Complications: allergic reactions and anaphylaxis, adverse events reported are frequent sequelae of open heart surgery (eg, atrial fibrillation, myocardial infarction, and heart failure).
Misc.: aprotinin prolongs whole blood clotting time of heparinized blood (prolonged PTT). Patients may require additional heparin even in the presence of activated clotting time (ACT) levels that appear to represent adequate anticoagulation. Standard loading dose of heparin should be employed, however, additional heparin should be administered in a fixed-dose regimen based on patient weight and duration of CPB. Patients undergoing deep hypothermic circulatory arrest have an increase incidence of renal failure and mortality. All doses of aprotinin should be administered through a central line alone (I.e., no other drugs should be administer in the same line).

Bretylium (Bretylol)
Actions: initially, release of norepinephrine into circulation, followed by prevention of synaptic release of norepinephrine; suppression of ventricular fibrillation and ventricular arrhythmias; increase in myocardial contractility (direct effect).
Indications: ventricular fibrillation and ventricular tachycardia.
Dose: 5 mg/kg IV push initially, followed by 5-10 mg/kg every 15-30 min to total of 30 mg/kg.
Continuous infusion: 1-2 mg/min (2 gms/500 cc D_5W at 30 cc/hr).
Complications: postural hypotension (potentiated by quinidine or procainamide), aggravation of digoxin induced arrhythmias, nausea/vomiting following rapid injection.

Calcium

Actions: essential for maintenance of cell membrane integrity, muscular excitation-contraction coupling, grandular stimulation-secretion coupling, and enzyme function.

Indications: hypocalcemia, hyperkalemia, hypomagnesemia, hypotension.

Dose: 500-1000 mg calcium chloride (2-10 mg/kg IV); 10% $CaCl_2$=1.36 mEq Ca^2/ml.

Adverse effects: may cause bradycardia or arrhythmia (especially with digitalis), hypertension, increased risk of ventricular fibrillation, and can be irritating to veins.

Dantrolene (Dantrium)

Actions: reduction of calcium release from sarcoplasmic reticulum.

Indications: malignant hyperthermia.

Dose: if signs of malignant hyperthermia develop: 3 mg/kg IV bolus; if syndrome persists after 30 minutes, repeat dose, up to 10 mg/kg (see section on malignant hyperthermia).

Complications: muscle weakness, gastrointestinal upset, drowsiness, sedation, or abnormal liver function.

Desmopressin Acetate (DDAVP)

Actions: synthetic product that increases plasma levels of Factor VIII and von Willebrand factor (vWF) and decreases bleeding times; causes release of tissue plasminogen activator and prostacyclin; antidiuretic activity.

Indications: bleeding uremic patients with platelet dysfunction, cirrhosis, cardiac surgery; improves coagulation in von Willebrand's disease and hemophilia; used as a antidiuretic hormone.

Dose: 0.3 mcg/kg IV over 20 minutes.

Complications: decreased free water clearance from antidiuretic activity, hypotension, thrombosis, decreased serum sodium.

Digoxin

Actions: (1) positive inotropic effects; (2) negative chronotropic effects; (3) slows conduction velocity through the AV node.

Pharmacokinetics: onset of action is about 30 min following IV and 30-120 min for oral tablets.

Indications: CHF, heart rate control in atrial fib/flutter.

Dose: (1) for supraventricular tachycardia: 10-15 mcg/kg IV in divided doses (0.25-0.5 mg as initial dose and 0.25 mg every 4 hours as subsequent doses until the entire dose is given or heart rate controlled); (2) CHF: 8-12 mcg/kg given in divided doses as above. All doses should be based on lean body weight.

Therapeutic level: 0.8-2.0 ng/ml.

Complications: symptoms of digoxin toxicity include mental depression, confusion, headaches, drowsiness, anorexia, nausea, vomiting, weakness, visual disturbances, delirium, EKG abnormalities (any arrhythmia) and seizures. Hypokalemia increases risk of digoxin toxicity. Treatment includes lidocaine or phenytoin for arrhythmias and Digibind for life threatening toxicity. Heart block potentiated by beta blockers or calcium channel blockers.

Diltiazem (Cardizem)

Actions: calcium channel antagonist; slows conduction through sinoatrial and AV nodes; dilates coronary and peripheral arterioles, and reduces myocardial contractility.

Indications: angina pectoris, variant angina from coronary artery spasm; temporary control of rapid ventricular rate during atrial fibrillation/flutter; conversion of paroxysmal supraventricular tachycardia to normal sinus rhythm.

Dose: (1) PO: 30-60 mg every 6 hours; (2) IV: initial bolus with 0.25 mg/kg over 2 minutes; if response is inadequate after 15 minutes, rebolus with 0.35 mg/kg over 2 minutes; for treatment give continuous infusion 5-15 mg/h (mix 125 mg in 100 cc of D_5W).

Complications: bradycardia and heart block; impaired contractility; transient increase in liver function tests. Adverse events include hypotension, injection site reaction, flushing, and arrhythmia.

Contraindicated in atrial fibrillation/flutter patients with WPW or short PR syndrone; sick sinus syndrome or second- or third-degree AV block except with a functioning pacemaker; severe hypotension or shock; hypersensitivity; recent IV beta-blockers; vent tachycardia.

Dobutamine (Dobutrex)

Action: beta 1 and beta 2 adrenergic agonist (greater increase in myocardial contractility than increase in heart rate); alpha 1 adrenergic agonist (beta 2 effect greater than alpha 1 effect; decreases systemic and pulmonary vascular resistance).

Indications: cardiogenic shock, severe CHF; low cardiac output.

Standard conc: 250 mg/250 D5W (1 mg/cc).

Dose: 2-20 mcg/kg/min. Drip rate: 15 cc/hr = 250 mcg/min.

Alternative preparation: 6 x body weight (kg) equals milligrams added to diluent (normal saline or D5W) to make 100 ml; then 1 cc/hr delivers 1.0 mcg/kg/min.

Complications: tachycardia (less than dopamine); minimal ventricular ectopy; hypertension.

Dopamine (Intropin)

Actions: Dopaminergic, alpha and beta adrenergic agonist

Indications: shock, poor perfusion, decreased splanchnic perfusion, low cardiac output.

Standard conc: 400 mg/250 cc D5W = 1600 mcg/cc.

Dosage range: 2-20 mcg/kg/min

2-5 mcg/kg/min: dopamine, min B1

5-10 mcg/kg/min: B1 > alpha

10-15 mcg/kg/min: alpha > B1

>15 mcg/kg/min: alpha agonist

Drip rate: 15 cc/hr = 400 mcg/min

Alternative preparation: 6 x body weight (kg) equals mgs added to diluent to make 100 ml; then 1 cc/hr delivers 1.0 mcg/kg/min.

Complications: tachycardia, arrhythmias, nausea/vomiting; superficial tissue necrosis and sloughing may occur with extravasation; use with dilantin may cause hypotension; contraindicated in pheochromocytoma, pulmonary hypertension may occur.

Dopexamine

Actions: synthetic analogue of dopamine, beta-2 and dopamine agonist (little beta-1 or alpha activity).

Indications:shock, poor perfusion, decreased splanchnic perfusion, low cardiac output, oliguria.

Dose:1-10 mcg/kg/min.

Complications: hypotension (causes vasodilation), tachycardia (atrial).

Droperidol

Actions: antiemetic effect, antipsychotic effect, apparent psychic indifference to environment, catatonia.

Dose: antiemetic: 0.625-2.5 mg.

Complications: evokes extrapyramidal reactions in 1%; possible dysphoric reactions; cerebral vasoconstrictor; can decrease blood pressure by alpha blockade and dopaminergic antagonism; used in neuroleptanalgesia.

Epinephrine

Actions: direct alpha and beta adrenergic receptors stimulation, resulting in bronchial smooth muscle relaxation, cardiac stimulation (positive inotrope), and dilation of skeletal muscle vasculature.

Standard conc: 2 mg/250 cc (15 cc/hr = 2 mcg/min).

Alternative preparation: 0.6 x body weight (kg) equals mgs added to diluent to make 100 ml; then 1 cc/hr delivers 0.1 mcg/kg/min.

Dose: start at 2 mcg/min (or 0.05 mcg/kg/min) and titrate to blood pressure and cardiac output; bolus starting at 10-20 mcg; infusion 0.01- 0.3 mcg/kg/min.

Ergonovine (Ergotrate)

Actions: constriction of uterine and vascular smooth muscle.

Indications: postpartum uterine atony and bleeding, uterine involution.

Dose: 0.2 mg IV in 5 ml NS given over 1 minute (IV route is used only in emergencies). 0.2 mg IM q2-4 hours for less than 5 doses; then PO: O.2-0.4 mg q6-12 hours for 2 days.

Complications: may cause hypertension from system vascoconstriction, arrhythmias, coronary spasm, cerebraovascular accidents, uterine tetany, or gastrointestinal upset.

Esmolol (Brevibloc)

Actions: selective beta-1 adrenergic blockade.

Indications: tachyarrhythmias, myocardial ischemia.

Dose: 50-300 mcg/kg/min.

Loading dose 500 mcg/kg bolus over 1 min followed by maintenance starting at 50 mcg/kg/min. To calculate an infusion rate in ml/min, divide mg/min by 10. To calculate an infusion rate in ml/hr, multiply mg/min by 6.

Standard conc: 10 mg/ml (two 2.5 gm ampuls in 500 cc).

Complications: bradycardia, AV conduction delay, hypotension, congestive heart failure.

Etomidate (Amidate)

Actions: augments the inhibitory tone of GABA in the CNS (produces unconsciousness in approximately 30 seconds).
Indications: IV induction agent for general anesthesia.
Induction: 0.2-0.3 mg/kg IV.
Maintenance: 10 mcg/kg/min IV with N 20 and opiate.
Sedation and analgesia: 5-8 mcg/kg/min; used only for short periods of time due to inhibition of corticosteroid synthesis.
Misc.: direct cerebral vasoconstrictor, minimal cardiovascular effects, pain on injection, myoclonus may occur in about 1/3 of pts during induction, adrenocortical suppression (dose dependent), nausea/vomiting.

Flumazenil (Romazicon)

Actions: competitive inhibition of GABA (benzo receptor in CNS).
Indications: reversal of benzodiazepine sedation or overdose.
Dose: (1) for reversal of conscious sedation: 0.2-1.0 mg IV every 20 minutes at 0.2 mg/min, (2) for overdose: 3-5 mg IV at 0.5 mg/min.
Complications: seizures, acute withdrawal, nausea, dizziness, agitation, arrhythmias, hypertension, resedation when reversing long-acting benzodiazepines.
Drug interactions: seizures in patients with prior seizure activity, tricyclic antidepressant poisoning, major hypnotic drug withdrawal.

Heparin

Actions: heparin, prepared from bovine lung, facilitates the activation of anti-thrombin III, neutralizes primarily thrombin and factor X.
Indications: anticoagulation.
Loading dose: (1) for thromboembolism: 5000 units IVP, (2) for cardiopulmonary bypass: 300 units/kg.
Maintenance dose: (1) for thromboembolism: start 1000 units/hr and titrate to PTT 1.5 to 2.5 x control, (2) for cardiopulmonary bypass: 100 units/kg/hr IV titrated against activated clotting time.
Standard conc: 10,000 units/500 cc D_5W (20 units per cc).
Reversal: reverse with protamine (see protamine).
Complications: hemorrhage, allergic reactions, thrombocytopenia, altered protein binding, cardiovascular changes (decreased MAP), decreased antithrombin III concentration, altered cell morphology, does not cross placenta.

Insulin

Indications: hyperglycemia.
Actions: facilitation of glucose transport into cells.
Infusion: 50 units regular insulin in 250 cc D_5W or NS (1 U/hr = 5 cc/hr).
Dose: average range is 2-10 units/hour or 0.1 units/kg/hour (see section on diabetes).
Complications: hypoglycemia, allergic reactions.
Misc: absorbed by plastic IV tubing.

Isoproterenol (Isuprel)

Actions: synthetic sympathomimetic amine that acts on beta-1 and beta-2 adrenergic receptors; positive chronotrope and inotrope; decreases systemic and pulmonary vascular resistance; increases coronary and renal blood flow; potent bronchodilator.

Indications: bradycardia, shock where increasing HR will increase CO, shock with severe aortic regurgitation, heart failure, pulmonary hypertension, refractory asthma, carotid sinus hypersensitivity.

Dose: 2-20 mcg/min; start at 1-2 mcg/min; peds: 0.01 mcg/kg/min.

Standard Conc: 1 mg/250 cc; 15 cc = 1 mcg.

Caution: don't use with in patients with myocardial infarction (may increase infarct size) or cardiac ischemia.

Complications: arrhythmogenic, especially with cardiac ischemia and digoxin toxicity; hypertension, CNS excitation, nausea/vomiting; pulmonary edema; paradoxical precipitation of Adams-Strokes attacks.

Ketamine

Induction

0.5-2 mg/kg IV; 4-6 mg/kg IM

Maintenance

0.5-1 mg/kg IV PRN with N 20 50%

15-45 mcg/kg/min IV with N 20 50-70%

30-90 mcg/kg/min IV without N 20

Sedation and analgesia

0.2-0.8 mg/kg IV; 2-4 mg/kg IM; 3 mg/kg Intranasally; 6-10 mg/kg PO

Misc: causes increased HR, CO, cardiac work, and myocardial oxygen requirements; direct stimulation of the CNS leads to increased sympathetic nervous system outflow (which may be blunted/prevented by prior administration of benzodiazepines or inhaled anesthetics). Ketamine itself is a direct myocardial depressant, potent cerebral vasodilator, and should not be used in patients receiving aminophylline (reduces seizure threshold).

Ketorolac (Toradol)

Action: inhibits prostaglandin synthesis through an effect on cyclo-oxygenase.

Indications: nonopioid, nonsteroidal analgesic for moderate pain.

Single dose (IM): patients <65 years of age: one dose of 60 mg; patients >65 years of age, renally impaired and/or less than 50 kg: one dose of 30 mg.

Single dose (IV): patients <65 years of age: one dose of 30 mg; patients >65 years of age, renally impaired and/or less than 50 kg: one dose of 15 mg.

Multiple dosing (IV or IM): patients <65 years of age: the recommended dose is 30 mg every 6 hours. The maximum daily dose should not exceed 120 mg. For patients >65 years of age, renally impaired and patients less than 50 kg should have one-half the above dose. The combined duration of use for parenteral and oral is not to exceed 5 days.

Complications: adverse effects are similar to those of other nonsteroidal anti-inflammatory drugs and include peptic ulceration. Contraindicated in patients with active peptic ulcer disease, advanced renal impairment, and risk of bleeding.

Misc.: potentiates the effects of opioids.

Lidocaine

Action: antiarrhythmic effect; sedation; neural blockade.

Loading Dose: 1-1.5 mg/kg IVP.

Maintenance: infusion 1-4 mg/min (reduce by half for pts with CHF, hepatic dysfunction, or shock).

Standard Conc: 2 gms/250 cc; (7 cc/hr = 1 mg/min).

Alternative Preparation: 120 mg of 40 mg/cc solution added to 97 cc of diluent, yielding 1200 mcg/cc solution; then 1 cc/hr delivers 20 mcg/kg/min.

Complications: CNS depression, drowsiness, unconsciousness, apprehension, change in vision, vomiting, bradycardia, hypotension, respiratory depression. Avoid in patients with Wolff-Parkinson-White syndrome.

Magnesium

Actions: central nervous system depressant and anticonvulsant, inhibits release of acetylcholine at the neuromuscular junction, decreases sensitivity of motor end-plate to acetylcholine, decreases muscle excitability, decreases uterine hyperactivity (thus increasing uterine blood flow), diminishes fibrin deposition, mild vasodilator and has a mild antihypertensive effect. Magnesium is excreted by kidneys. Hypermagnesemia can result in a drowsy neonate with decreased muscle tone and hypoventilation.

Indications: pregnancy induced hypertension, hypomagnesemia.

Dose: (1) for treatment of hypomagnesemia: 4-8 gms/100 cc NS or D_5W given over 8 hours; (2) for pregnancy induced hypertension patients: initial bolus of 2-4 gms in a 20% solution IV over 5 minutes followed by a continuous infusion of 1-2.5 gms/hr (10-20 gms in 1000 cc of D_5W).

Levels: normal plasma level is 1.5 to 2.2 mEq/l. In the treatment of pregnancy induced hypertension the therapeutic level is 4-5 mEq/l.

Complications:(1) at 5-10 mEq/l: there is loss of deep tendon reflexes and respiratory depression; (2) at 10 mEq/l: there are prolonged PR and QT intervals and widened QRS complexes; (3) at 15 mEq/l: there is SA and AV nodal blocks and respiratory apnea; (4) at 25 mEq/l: cardiac arrest can occur.

Mannitol (Osmitrol)

Actions: increase in serum osmolality resulting in decrease in brain size and amount of intraocular fluid, osmotic diuresis, and transient expansion of intravascular volume.

Indications: Intracranial hypertension, prophylaxis/treatment of renal failure, glaucoma, diuresis.

Dosage: 0.25-1.0 gm/kg IV as 20% solution over 30-60 minutes.

Complications: rapid administration may cause vasodilation and hypotension. May cause pulmonary edema, intracranial hemorrhage, systemic hypertension.

Methohexital (Brevital)
Indications: IV induction agent for general anesthesia.
Induction: 1-1.5 mg/kg IV or 20-30 mg/kg pr (children).
Sedation: 0.2-0.4 mg/kg IV.
Misc: earlier recovery time and less cumulative effect than thiopental, higher incidence of excitatory phenomena (cough, hiccups, involuntary movements), pain on injection, activates epileptic foci (unlike other barbiturates).

Methylergonovine (Methergine)
Actions: constriction of uterine and vascular smooth muscle.
Indications: postpartum hemorrhage.
Dose: 0.2 mg IV in 5 ml NS given over 1 minute (IV route is used only in emergencies). 0.2 mg IM q2-4 hours for less than 5 doses; then PO: O.2-0.4 mg q6-12 hours for 2 days.
Complications: may cause hypertension from system vasoconstriction, arrhythmias, coronary spasm, uterine tetany, or gastrointestinal upset.

Milrinone (Primacor)
Indications: low output heart failure.
Actions: phosphodiesterase inhibitor, increases cAMP in the heart, peripheral vasodilator, increases inotropy, approximately 20 times the inotropic potency of amrinone.
Loading dose: 50 mcg/kg IV over 10 minutes.
Maintenance dose: 0.375-0.75 mcg/kg/min.
Standard concen: 200 mcg/cc (50 mg/250 cc dextrose or saline).
Complications: increased ventricular ectopy, including nonsustained ventricular tachycardia, supraventricular tachycardia; hypotension; headaches.
Misc.: the presence of renal impairment may significantly increase the terminal elimination half-life. Similar to amrinone, but does not cause fever or thrombocytopenia. Do not inject furosemide into intravenous lines containing milrinone; a precipitate-forming chemical reaction will occur.

Naloxone (Narcan)
Indications: reversal of systemic narcotic effects.
Actions: antagonism of narcotic effect.
Dose:0.04-0.4 mg IV titrated against patient response q2-3 minutes; Pediatric: 1-10 mcg/kg q2-3 minutes up to 0.4 mg. May be given as continuous infusion 5-10 mcg/kg/hr
Onset/duration: onset 1-2 minutes; duration less than one hour.
Misc: may cause reversal of analgesia, hypertension, arrhythmias, rare pulmonary edema, delirium or withdrawal syndrome.

Nicardipine (Cardene)
Actions: dihydropyridine calcium channel blocker.
Indications: for short-term treatment of hypertension.
Dose:administer by slow continuous infusion. Initiate therapy at 50 cc/hr (5.0 mg/hr). If desired blood pressure reduction is not achieved at this dose, the infusion rate may be increased by 25 cc/hr every 5 minutes for rapid blood pressure reduction or every 15 minutes for gradual blood pressure reduction up to a maximum of 150 cc/hr. Following

achievement of the blood pressure goal, the infusion rate should be decreased to 30 cc/hr.

Contraindications: patients with known hypersensitivity to the drug and those with advanced aortic stenosis. Caution is advised when administering in patients with impaired renal or hepatic function or in combination with a beta blocker in CHF patients.

Side effects: most commonly hypotension, headache, tachycardia, and nausea/vomiting.

Transfer to oral nicardipine: administer first dose of TID regimen 1 hour prior to discontinuation of continuous infusion.

Infusion rate	Equivalent oral dose
0.5 mg/hr	20 mg q8 hrs
1.2 mg/hr	30 mg q8 hrs
2.2 mg/hr	40 mg q8 hrs

Nitroglycerin

Actions: smooth muscle relaxant; greater venous dilation than arterial dilation (decrease of preload > decrease of afterload), coronary artery dilation, decreased systemic vascular resistance, decreased pulmonary vascular resistance.

Indications: myocardial ischemia, hypertension, congestive heart failure, pulmonary hypertension, esophageal spasm, biliary colic.

Dose: start at 5-10 mcg/min and titrate and advance every 5 minutes until CP resolved or hemodynamic state achieved.

Standard Conc: 50 mg/250 cc; 3 cc/hr = 10 mcg/min.

Complications: reflex tachycardia, hypotension, headache, methemoglobinemia with high doses. Tolerance and dependence with chronic use. May be absorbed by plastic in IV tubing.

Nitroprusside

Actions: smooth muscle relaxation; arterial dilation greater than venous.

Indications: hypertension, induction of deliberate hypotension, congestive heart failure, pulmonary hypertension.

Dose: 0.5-10 mcg/kg/min; start at 20 mcg/min.

Standard Conc: 50 mg/250 cc D_5W (1 cc = 200 mcg); 15 cc/hr = 50 mcg/min.

Complications: hypotension, nausea, headaches, restlessness, cyanide and thiocyanate toxicity, degraded by light (tubing/container must be covered with aluminum foil), signs of toxicity include tachyphylaxis, metabolic acidosis, and high mixed venous saturation (treatment is with sodium nitrite, sodium thiosulfate, hydroxyocobalamin or methylene blue).

Misc.: nitroprusside enters red blood cells where it receives an electron from the iron of oxyhemoglobin and eventually results in methemoglobin and 5 cyanide ions. Cyanide ions undergo one of three possible reactions: binding to methemoglobin to form cyanmethemoglobin; undergoing a reaction in the liver and kidney by the enzyme rhodanase; or binding to tissue cytochrome oxidase.

Norepinephrine (Levophed)
Actions: alpha 1, alpha 2, and beta 1 adrenergic agonist.
Indications: shock with sepsis, decreased SVR and elevated CO.
Dose: 1-20 mcg/min.
Standard Conc: 4 mg/250 cc; 15 cc/hr = 4 mcg/min.
Complications: ischemic necrosis and sloughing of superficial tissues will result if extravasation occurs, hypertension, arrhythmias, myocardial ischemia, increased uterine contractility, constricted microcirculation, CNS stimulation.

Ondansetron (Zofran)
Actions: serotonin receptor selective antiemetic.
Indications: prevention of postoperative nausea and/or vomiting.
Adult Dose: 4 mg undiluted IV in not less than 30 seconds.
Pediatric Dose: 0.05-0.075 mg/kg IV in not less than 30 seconds.
Complications: headache, dizziness, musculoskeletal pain, drowsiness, sedation, shivers.

Phenylephrine (Neo-Synephrine)
Actions: alpha adrenergic agonist.
Indications: hypotension.
Dose: 50-200 mcg bolus or 20-100 mcg/min continuous infusion.
Standard conc: for bolus 50 mcg/cc; for infusion 40-100 mcg/cc.
Complications: hypertension, reflex bradycardia, constricted microcirculation, uterine contraction.

Procainamide (Pronestyl)
Actions: antiarrhythmic effect.
Indications: atrial and ventricular arrhythmias.
Load: continuous infusion of 20-30 mg/min until (1) arrhythmia suppressed; (2) hypotension ensues; (3) the QRS complex widened by 50%; or (4) a total of 1 gm or 17 mg/kg has been given.
Maintenance: 1-4 mg/min.
Standard Conc: 2 gms/500 cc D_5W: 30 cc/hr = 2 mg/min.
Therapeutic level: 4-10 mcg/ml.
Complications: hypotension, heart block, myocardial depression, ventricular dysrhythmias, lupus, fever, agranulocytosis, GI irritation, CNS symptoms.

Propofol
Mechanism: involves facilitation of the inhibitory neurotransmission mediated by gamma aminobutyric acid.
Indications: IV induction agent for general anesthesia.
Induction: 1.5-2.5 mg/kg; reduce w/age >50.
Maintenance: 0.1-0.2 mg/kg/min or 6-12 mg/kg/hr combined with N_20 and opiate.
Sedation: 25-150 mcg/kg/min.
Misc: propofol contains 0.005% disodium edetate, which has been found to retard the rate of growth of microorganisms in the event of accidental extrinsic contamination.

Prostaglandin E1 (Alprostadil)

Actions: vasodilation, inhibition of platelet aggregation, vascular smooth muscle relaxation, uterine and intestinal smooth muscle relaxation.

Indications: pulmonary vasodilator, maintenance of patent ductus arteriosus.

Standard Conc: 500 mcg (1 vial) in 99 cc D_5W (5 mcg/cc).

Dose: start at 0.05 mcg/kg/min (0.01 cc/kg/min); may increase to as high as 0.5 mcg/kg/min.

Complications: hypotension, apnea, flushing, bradycardia.

Misc.: rapidly metabolized (approx. 70% metabolized in one passage through the lungs).

Protamine

Actions: specific antagonist of heparin's anticoagulant effect. Protamine is found in the sperm and testes of certain fish. Protamine administered in the absence of heparin may interact with platelets and proteins manifesting as an anticoagulant effect.

Indications: reversal of the effects of heparin.

Dose: 1-3 mg per 100 units heparin, maximum 50 mg over 10 minutes, given slowly. A guideline administration of 1.3 mg/kg of protamine for each 100 units of heparin present as calculated from the ACT.

Complications: Hypotension (seen with rapid injection secondary to histamine release), pulmonary hypertension (protamine neutralization of heparin can result in complement activation and thromboxane release manifesting as pulmonary hypertension), and allergic reactions (seem in patients receiving protamine containing insulin preparations and in some patients allergic to fish).

Vasopressin (Pitressin)

Actions: synthetic analogue of arginine vasopressin; antidiuretic, and produces contraction of the smooth muscle of the GI tract and vascular bed.

Indications: diabetes insipidus, GI bleed, hemophilia, control of post operative ileus.

Standard Conc: 200 units vasopressin in 250 cc D_5W or NS (0.8 units/ml)

Dose: diabetes insipidus: 5-10 units SC/IM bid or tid prn; GI hemorrhage: infusion of 0.2-0.9 units/min

Complications: cardiotoxicity (myocardial ischemia, bradycardia), abdominal cramps, nausea, diarrhea, bowel infarction, water intoxication.

Misc: usually combined with nitroglycerin infusion to reverse cardiotoxic effects while reducing the increase in portal venous resistance.

Drip Rates

cc/hr = (mcg/kg/min x 60 x wt(kg))/mcg/cc

mcg/kg/min = [(cc/hr) x (mcg/cc)]/60 x wt(kg)

Parenteral Agents for the Acute Treatment of Hypertension

Agent	Dosage Range	Onset	Duration
Nitroprusside	0.5-10 mcg/kg/min	30-60 sec	1-5 min
Nitroglycerin	5-100 mcg/min	1 min	3-5 min
Esmolol	0.5 mg/kg over 1 min 50-300 mcg/kg/min	1 min	12-20 min
Labetalol	5-20 mg	1-2 min	4-8 hr
Propranolol	1-3 mg	1-2 min	4-6 hr
Trimethaphan	3-4 mg/min	1-3 min	10-30 min
Phentolamine	2.5-5 mg	1-10 min	20-40 min
Diazoxide	1-3 mg/kg slowly	2-10 min	4-6 hr
Hydralazine	5-20 mg	5-20 min	4-8 hr
Nifedipine (SL)	10 mg	5-10 min	4 hr
Nicardipine	5-15 mg/hr	1-5 min	3-6 hr
Methyldopa	250-1000 mg	2-3 hr	6-12 hr

Pharmacology of Antihypertensive Parenteral Agents

Drug	Site of Vasodilation	Advantages	Side Effects/Problems
Nitroprusside	Direct dilator (balanced)	Immediate action Easy to titrate No CNS effects	Hypotension Reflex tach Cyanide toxicity Methemoglobin Avoid in renal failure
Nitroglycerin	Direct dilator (venous > arterial	Coronary dilator	Headache Absorbed into IV tubing ETOH vehicle
Hydralazine	Direct dilator (arterial >> venous	No CNS effects	Reflex tach, Lupus, Local thrombophlebitis
Diazoxide	Direct dilator (arterial >> venous	Rapid action Not sedating	Imprecise dosing Reflex tach Hyperglycemia Hyperuricemia
Trimethaphan	Ganglionic blocker (balanced)	Aortic aneurysm Subarachnoid bleed	Anticholinergic effects Decreased cardiac output
Phentolamine	Alpha blocker and direct vasodilator	Pheochromo-cytoma MAO crisis	Reflex tachycardia Tachyphylaxis
Labetalol	Alpha and beta blockers	No overshoot hypotension Maintained CO, HR	Exacerbation of CHF, asthma, AV block, bronchospasm

Classification of Antiarrhythmic Agents

Class	Mechanism of Action	Agents
I	Blocks fast sodium channels (decrease slope of phase O)	
Ia	Dissociation intermediate, conduction moderately depressed, repolarization accelerated	Quinidine Procainamide Disopyramide
Ib	Dissociation rapid, conduction minimally depressed, repolarization accelerated	Lidocaine Phenytoin Tocainide Mexiletine
Ic	Dissociation slow, conduction markedly depressed, no effect on repolarization	Flecainide Encainide
II	Block beta-adrenergic receptors (decreases automaticity)	Propranolol Esmolol
III	Repolarization prolonged	Amiodarone Bretylium Sotalol
IV	Block slow calcium channels (decrease automaticity and conduction in nodal tissue)	Verapamil

Cardiovascular Physiology and Anesthesia

Cardiopulmonary /Hemodynamic Parameters

Parameter	Formula	Normal Range
RA, CVP		2-10 mmHg
LA or LVEDP		4-12
RV		15-30/0-5 mmHg
PAS/ PAD		15-30/8-15 mmHg
MPAP	PAD + (PAS-PAD/3) or [(PAD)2 + PAS]/3	10-18 mmHg
PCWP		5-16 mmHg
MAP	DBP + (SBP-DBP/3) or [(DBP)2 + SBP]/3	75-110 mmHg
SVR	(MAP-CVP x 79.9)/CO	770-1500 dynes/sec/cm5
PVR	(MPAP-PCWP x 79.9)/CO	20-120 dynes/sec/cm5
$Ca0_2$	(Hgbx1.34)SaO_2+(PaO_2x 0.0031)	16-22 mls O_2/dl blood
CvO_2	(Hgbx1.34)SvO_2+(PaO_2x 0.0031)	12-77 ml O_2/dl blood
$C(a\text{-}v)O_2$	(Hgb x 1.34)(SaO_2 - SvO_2)	3.5-5.5 ml O_2/dl blood
SV	CO x 1000/HR	60-100 ml
CO	SV x HR=VO_2/$C(a\text{-}v)O_2$x10	3-9 liters/min
CI	CO/body surface area	2.1-4.9 L/min/m2
DO_2	CaO_2 x CO x 10	700-1400 ml/min

Cardiopulmonary /Hemodynamic Parameters Continued

Parameter	Formula	Normal Range
PAO_2	FIO_2 x (PB-PH_2O)-($PaCO_2$/0.8)	
A-a gradient	[(713)FIO_2-$PaCO_2$ (1.25)]-PaO_2	2-22 mmHg (RA)
Qs/Qt	(CcO_2-CaO_2)/(CcO_2-CvO_2)	0.05 or less
Creatinine Clearance	(Urine Cr/Serum Cr) x 70	Male=125; Female=105
Fract Exc of Sodium	(Ur Na/Plasma Na)/(Ur Cr/Pl Cr) x 100	pre-renal <1; ATN>1
PaO_2	102-(age/3)	
Coronary Perfusion Pressure (CPP)	Art Diastolic BP - LVEDP	
SvO_2	SaO_2-VO_2/(CO x Hb)(13.4)	68-77%
VO_2 (approximate)	Hb x 1.34 x CO x 10 x (SaO_2-SvO_2)	
Fick equation (VO_2)	CO x C(a-v)O_2	225-275 ml/min
Body Mass Index (BMI)	wt (kg)/ht $(m)^2$	Obese >28 Morbidly obese >35
V_D/V_T	(PaCO2-P_ECO_2)/$PaCO_2$	Normal: 33%.

Cardiovascular Agents: Systemic Effects

Drug	CO	PCWP	SVR	MAP	HR	CVP	PVR
Norepinephrine	I	I	I	I	NC	I	I
Phenylephrine	D	I	I	I	D	I	I
Epinephrine	I	I	I	I	I	I	I
Dobutamine	II	D	D	I	I	D	D
Dopamine <6 mcg/kg/min	I	I	I	I	I	I	NC
Dopamine >6 mcg/kg/min	I	II	II	II	I	II	I
Digoxin	I	NC	NC	NC	D	NC	NC
Isoproterenol	II	D	D	D	II	D	D
Amrinone	I	D	D	NC	I	D	D
Nitroglycerin 20-40 mcg/min	NC	D	NC	NC	NC	D	NC
Nitroglycerin 50-250 mcg/min	I	D	D	D	I	D	D
Nitroprusside	I	D	D	D	I	D	D

Key: I=Increased; II=Large Increase; D=Decreased; NC=No Change

Adrenergic Agonists: System Effects

Drug	HR	MAP	CO	PVR	Bronchodilation	Renal BF
Phenylephrine	-	+++	-	+++	0	---
Methyldopa	-	--	-	--	0	+
Epinephrine	++	+	++	-/+	++	--
Ephedrine	++	++	++	+	++	--
Norepinephrine	-	+++	-/+	+++	0	---
Dopamine	+/++	+	+++	+	0	+++
Isoproterenol	+++	-	+++	--	+++	-/+
Dobutamine	+	+	+++	-	0	+

Key: O = No effect; + = Minimal effect; ++ = Moderate effect; +++ = Marked effect. HR = heart rate; MAP = mean arterial pressure; CO = cardiac output; PVR = pulmonary vascular resistance; BF = blood flow.

Adrenergic Agonists: Receptor Selectivity

Drug	Alpha 1	Alpha 2	Beta 1	Beta 2	Classification
Methoxamine	+++	+	0	0	
Phenylephrine	+++	+	+ (hi doses)	0	D
Metaraminol	+++	?	++	0	I
Methyldopa	+	+++	0	0	
Clonidine	+	+++	0	0	D
Epinephrine	++	++	+++	++	D
Ephedrine	++	?	++	+	I
Norepinephrine	++	++	++	0	D
Dopamine	++	++	++	+	M
Mephentermine	+	?	++	+	I
Isoproterenol	0	0	+++	+++	D
Dobutamine	0/+	0	+++	+	
Terbutaline	0	0	+	+++	

Key: 0 = no effect; + = agonist effect (+ = mild, ++ = moderate, +++ = marked); ? = unknown effect; D = direct; I = indirect; M = mixed.

Electrocardiograms

Interpretation Format **Normal Intervals (each small block=0.04 sec)**

1. Rate — P-R: 0.12-0.20 msec
2. Rhythm — QRS: 0.06-0.10 msec
3. Intervals
4. Axis
5. Hypertrophy
6. Infarction/ischemia
7. Ectopy

Q-T (msec)	HR (bpm)
0.33-0.43	60
0.31-0.41	70
0.29-0.38	80
0.28-0.36	90
0.27-0.35	100

Electrolyte Abnormalities On EKG

Hyperkalemia
- tall peaked T waves
- QRS widening
- ST elevation
- loss of P waves

Hypokalemia
- small T wave/U wave
- QRS widening
- ST depression

Hypercalcemia
- shortening of QTc

Hypocalcemia
- prolongation of QTc

Hyponatremia
- no change

Hypernatremia
- no change

Localization Of Myocardial Infarction

Location of MI	Q wave or ST Change	Reciprocal ST Depress
Anterior	V2-V4 (also poor R wave progression V1-V6)	II, III, AVF
Antero-Septal	V1-V3	
Antero-Lateral	I, aVL, V4-V6	
Lateral	I, AVL, V5-V6	V1, V3
Inferior	II, III, AVF	I, AVL
Posterior	Tall R/T waves V1-V3	V1-V3
Subendocardial	ST depression in ant leads or inferior leads	

(Q wave >0.04 sec and >25% the height of the R wave)

LVH Criteria
- RV5 or RV6 >26 mm
- SV1 or SV2 + RV5 or RV6 >35 mm
- R I +S III >25 mm
- RaVL >13 mm
- RaVF >20 mm

RVH Criteria
- R>S in V1 or V3
- qR in V1 or V3
- RAD
- Wide QRS

Atrial Hypertrophy

RAH: diphasic P with tall initial component

LAH: diphasic P with persistent S in V4-V6 wide terminal component

Bundle Branch Block (QRS >0.12 sec)

Left BBB
- Absence of septal Q waves in V4-V6, I, AVL
- RR' or M pattern of QRS in I, aVL, V4-V6
- 2° ST, T wave change I, aVL, V4-V6

Right BBB
- RR' or M pattern of QRS in V1-V3
- Deep/round S waves in I, aVL, V4-V6
- 2° ST, T wave change in V1-V3

AV Heart Blocks

1°=PR interval >0.20 sec.

2°= Mobitz Type I (Wenckebach): PR interval increases until QRS dropped, delta wave.

Mobitz Type II (infra His dysfunction) PR interval constant until QRS dropped.

3°=No AV conduction (P has no relation to QRS).

EKG Changes

Prolonged QT Interval: hypocalcemia, hypokalemia, hypomagnesemia, acute MI, acute myocarditis, procainamide, quinidine, tricyclic antidepressants, subarachnoid hemorrhage, Jervell-Lange-Nielsen syndrome, Romano-Ward, right radical neck dissection.

Shortened QT Interval: hypercalcemia, digitalis.

LAD: LVH, left ant hemiblock, inferior wall MI.

RAD: RVH, left post hemiblock, dextrocardia, pulmonary infarct, RBBB, lateral MI.

Pericarditis: diffuse ST elevation concave upward and/or diffuse PR depression and/or diffuse T wave inversion.

Digitalis toxicity: ventricular arrhythmias, conduction abnormalities.

Quinidine/procainamide: prolonged QT, flattened T wave, QRS widening.

Hypothermia: bradycardia, AV junctional, elevated J point, prolong QT.

Orthotopic heart transplantation: the patient's original SA node often remains with the original atria, therefore, two P waves can be seen.

Factors Favoring PVC's versus Supraventricular Aberrancy

Monophasic or diphasic complex in lead V1

Notched QRS complex with R > R'

QS in V6 or an R/S ratio in V6 <1.0

Absent P waves or PR interval < 0.10 seconds

QRS duration > 0.14

Fully compensatory pause

Fusion beat

Electrocardiographic Detection of Perioperative Myocardial Ischemia

Single lead EKG sensitivity: V5 (75%), V4 (61%), V6 (37%), V3 (33%), II (24%), and all others <14%.

Combination leads: leads II and V5 increase sensitivity to 85%, leads V4 and V5 increase sensitivity to 90%, increasing to 96% by combining II, V4, and V5, and to 100% when five leads were used (V2-V5 and II).

Pacemakers: Nomenclature for Description of Pacemaker Function

First Letter: Chamber Paced	Second Letter: Chamber Sensed	Third Letter: Generator Response	Fourth Letter: Program Functions	Fifth Letter: Antitachy-cardia Functions
V-Ventricle	V-Ventricle	T-Triggered	P-Program[2]	B-Bursts
A-Atrium	A-Atrium	I-Inhibited	M-Multi-program	N-Normal rate competition[4]
D-Dual chamber	D-Dual chamber	D-Dual chamber	C-Communi-cating	S-Scanning[5]
	O-None (Asyn-chronous)	O-None (Asyn-chronous)	O-None (fixed function)	E-External[6]
		R-Reverse Functions[1]		

1= Pacemaker activated at fast rates only (i.e., upon sensing a tachyarrhythmia as opposed to bradyarrhythmia).
2 = Rate and/or output only.
3= Telemetry, interrogation (P or M implicit).
4 = Paces at normal rate upon sensing tacharrhythmia (underdrive pacing).
5 = Scanning response (such as time extrasystoles).
6 = External control (activated by a magnet, radiofrequency, or other means).

1. Preoperative pacemaker evaluation

A. Determine the reason for the pacemaker, the type of generator, the date and pulse the generator was placed, and the preset rate.

B. Determine pacemaker function

1. The rate of discharge of asynchronous pacemakers is the most important indicator of pulse generator function.
2. A 10 percent decrease in heart rate from the initial fixed discharge rate is a sign of battery failure.
3. Electrocardiogram to evaluate one-to-one capture. This is tested by monitoring the electrocardiogram while simultaneously palpating a peripheral pulse (each paced beat should correspond to a pulse). This test, however, is not helpful in patients with VVI pulse generators with heart rates greater than the preset pacemaker rate, ventricular synchronous or sequential cardiac pacemakers. In these patients proper function can be confirmed by demonstration of captured beats on the electrocardiogram when the pacemaker is converted to the asynchronous mode by placement of an external converter magnet over the pulse generator (this may not work for newer pacers).
4. If the generators is less than 2 years old, does not produce pacing impulses when the patient's heart rate is above 72 beats per min, and does produce pacing impulses

associated with a peripheral pulse when the heart rate slows, generally can be considered properly functioning.

C. The patient should be questioned for history of vertigo, syncope, light headedness, or return of any pre-pacemaker symptoms which may reflect dysfunction of the pacemaker.

D. Laboratory: serum electrolytes (hypokalemia can increase the negative cell membrane potential increasing threshold for pacemaker to capture).

E. Chest x-ray: looking for a dislodged electrode or fracture, and the make and model, if available.

2. Pacemaker failure

A. Acute potassium changes.

1. Decreased extracellular potassium: can result in loss of pacing
2. Increased extracellular potassium: caused by myocardial ischemia, depolarizing muscle relaxants or rapid potassium replacement all can result in pacing-related ventricular fibrillation.

B. Myocardial infarction can result loss of pacing.

C. Electromagnetic interference can result in loss of pacing or reprogramming.

3. Indications for temporary pacemaker

A. Symptomatic sick sinus syndrome.

B. Symptomatic hypertensive carotid sinus syndrome with cardio-inhibitory (not just vasodepressor) response.

C. Mobitz type 2 block.

D. Acute MI with RBBB or LBBB.

E. Comatose trauma patient with bifascicular block.

F. Trifascicular block.

G. Symptomatic beta blocker overdose.

Central Pressure Monitoring: Central Venous Pressure Waves

Component	EKG	Cardiac Cycle	Mechanical Event
a Wave	Follows P wave	End diastole	Atrial Contraction
c Wave	Follows the onset of the QRS	Early systole	Isovolumic ventricular contraction, tricuspid motion toward atrium
v Wave	Follows T wave	Late systole	Systolic filling of atrium
h Wave		Mid diastole	Mid-diastolic pressure plateau (occurs with slow heart rates and prolonged diastole)
x Descent		Midsystole	Atrial relaxation, descent of the base, systolic collapse
y Descent		Early diastole	Early ventricular filling, diastolic collapse

Central Venous Pressure Monitoring

1. **Indications for central venous pressure monitoring**
   - A. Major thoracic procedures involving large fluid shifts or blood losses in patients with good heart function.
   - B. Intravascular volume assessment when urine output is unreliable or unavailable (eg, renal failure).
   - C. Major trauma.
   - D. Frequent blood sampling in patients who will not require an arterial line.
   - E. Venous access for vasoactive or irritating drugs.
   - F. Chronic drug administration
   - G. Inadequate peripheral intravenous access.
   - H. Rapid infusion of intravenous fluids using large cannulae.
2. **Respiratory influences**
   - **A. End expiration:** CVP measurements should be made at end expiration, because pleural and pericardial pressures approach atmospheric pressure under these conditions, regardless of whether the patient is breathing spontaneously or receiving positive-pressure mechanical ventilation.
     1. **Spontaneous ventilation:** during spontaneous breathing, inspiration causes a decrease in intrapleural pressure and juxtacardiac pressure, which is transmitted in part to the right atrium and produces a decrease in CVP.
     2. **Mechanical ventilation:** positive-pressure ventilation causes intrathoracic and juxtacardiac pressure to increase during

inspiration, producing a increase in CVP.

B. PEEP: interpretation of the CVP with added PEEP or CPAP can be difficult. Clearly, as intrathoracic pressure increases from added PEEP, CVP measurements increases. However, this may be associated with a reduction in transmural filling pressure, preload, and venous return.

3. CVP Abnormalities

A. Atrial fibrillation: the a wave disapprears, and the c wave becomes more prominent since atrial volume is greater at end-distole. Fibrillation waves may be noticed in the CVP trace in some patients.

B. Isorhythmic A-V dissociation or junctional rhythm: atrial contraction may occur against a closed tricuspid valve, resulting in a "cannon a wave."

C. Tricuspid regurgitation: causes "ventricularization" of the CVP trace, with a broad, tall systolic c-v wave that begins early in systole and obliterates the x descent. Unlike a normal v wave, the c-v wave begins immediately after the QRS, leaving only a y descent.

D. Tricuspit stenosis: prominent a wave as the atrium contracts against a stenotic valve; the y descent following the v wave is obstructed.

E. Right ventricular ischemia and infarction

1. Diagnosis suggested by arterial hypotension in combination with disproportionate elevation of the CVP as compared to the PCWP. Mean CVP may approach or exceed the mean PCWP.
2. Elevated right ventricular filling pressure produces prominent a and v waves and steep x and y descents, giving the waveform an M or W configuration

F. Pericardial constriction: central venous pressure is usually markedly elevated, and the trace resembles that seen with right ventricular infarction: prominent a and v waves and steep x and y descents, creating an M pattern. Often the steep y descent in early diastole is short lived, and the CVP in mid-distole rises to a plateau until the a wave is inscribed at end-diastole (similar to the h wave).

G. Cardiac tamponade: venous pressure waveform becomes monophasic with a characteristic obliteration of the diastolic y descent. The y descent is obliterated because early diastolic runoff from atrium to ventricle is impaired by the compressive pericardial fluid collection.

4. Contraindications to central venous pressure monitoring

A. Absolute contraindications

1. Superior vena cava syndrome.

B. Relative contraindications

1. Infection at the site of insertion.
2. Coagulopathy.
3. Newly inserted pacemaker wires.

Pulmonary Artery Catheter Distances (cm)

Vein	Right Atrium	Right Ventricle	Pulmonary Artery
Internal Jugular			
Right	20	30	40
Left	25	35	50
Subclavian	15	25	40
Antecubital			
Right	40	50	65
Left	45	55	70
Femoral	30	40	55

Pulmonary Artery Catheterization

1. **Common indications for pulmonary artery catheterization**
   - A. Cardiac disease
     1. Cardiac disease with left ventricular dysfunction or recent myocardial infarction.
     2. Valvular heart disease.
     3. Heart failure.
   - B. Pulmonary disease
     1. Acute respiratory failure.
     2. Severe chronic obstructive pulmonary disease.
   - C. Complex fluid management.
   - D. Specific surgical procedures.
2. **Contraindications**
   - A. Absolute contraindications
     1. Tricuspid or pulmonic valvular stenosis.
     2. Right atrial or right ventricular massess (eg, tumor, clot).
     3. Tetralogy or Fallot.
   - B. Relative contraindications
     1. Severe dysrhythmias: complete left bundle branch block (because of the risk of complete heart block), Wolff-Parkinson-White syndrome, and Ebstein's malformation (because of possible tachyarrhythmias).
     2. Coagulopathy.
     3. Newly inserted pacemaker wires.
3. **Correct position** can be confirmed by a chest radiograph. Although most catheters migrate caudally and to the right side, occasionally a catheter will wedge anterior to the vena cava. In this position, true pulmonary capillary pressures may be less than alveolar pressures, resulting in spuriously elevated measurements during positive pressure ventilation.
4. **Indiators of proper tip placement include**
   - A. A decline in pressure as the catheter moves from the pulmonary artery into the "wedged" position.
   - B. Ability to aspirate blood from the distal port (eliminating the possibility of overwedging).
   - C. A decline in end-tidal CO2 concentration with inflation of the balloon (prodcued by a rise in alveolar dead space).
5. **Complications**
   - A. Endobronchial hemorrhage. The incidence of pulmonary artery catheter-induced endobronchial hemorrhage is 0.06%-0.20%. Risk factors include advanced age, female sex, pulmonary hypertension, mitral stenosis, coagulopathy, distal placement of the catheter, and balloon hyperinflation.
   - B. Pulmonary infarction.

Swan-Ganz Catheter in the Diagnosis of Low Cardiac Output

Etiology of Low Cardiac Output	RA or CVP	PCWP	PAD vs PCWP
Hypovolemia	low	low	PADP = PCWP
Left ventricular failure	normal or high	high	PADP = PCWP
Right ventricular failure	high	normal	PADP = PCWP
Pulmonary embolism	high	normal	PADP > PCWP
Chronic pulm HTN	high	normal	PADP > PCWP
Cardiac tamponade	high	high	PADP = PCWP

Conditions Resulting in Discrepancy Between PCWP And LVEDP

1. **Overestimating (PCWP > LVEDP)**
   - A. Positive pressure ventilation
   - B. PEEP
   - C. Increased intrathoracic pressure
   - D. Non-zone III PAC placement
   - E. COPD
   - F. Tachycardia
   - G. Increased pulmonary vascular resistance
   - H. Mitral valve obstruction (stenosis, myxoma, clot)
   - I. Pulmonary venous compression (tumor, fibrosis)
   - J. Mitral regurgitation
   - K. Left-to-right intracardiac shunt
2. **Underestimating (PCWP < LVEDP)**
   - A. Noncompliant left ventricle
   - B. Aortic regurgitation
   - C. Reduced pulmonary arterial tree (pneumonectomy, PE)

Chest Radiography (CXR)

1. **Endotracheal tube position**
   A. When the head is in the neutral position, the tip of the ET tube should rest in the mid-trachea, approximately 5 cm above the carina. In adult patients the T5-T7 vertebral level is a good estimate of carinal position if it cannot be directly visualized.
2. **Central venous catheters**
   A. The desirable location is in the mid-superior vena cava, with the tip directed inferiorly.
   B. The catheter's tip should not be allowed to migrate into the heart cambers.
3. **Pulmonary artery catheters**
   A. The tip of the pulmonary artery catheter should rest below the level of the left atrium (zone 3) to reduce or eliminate transmission of alveolar pressure to the capillaries.
   B. With an uniflated balloon, the tip ot the pulmonary artery catheter should overlie the middle third of a well-centered AP chest x-ray (within 5 cm of the midline). Distal migration is common in the first hours after insertion as the catheter softens and loses slack.
4. **Intra-aortic ballon pump (IABP)**
   A. Diastolic inflation of the balloon produces a distinct, rounded lucency within the aortic shadow, but in systole the deflated balloon is not visable. Ideal positioning places the catheter tip just distal to the left subclavian artery.

Factors Affecting Myocardial Oxygen Supply-Demand

Supply	**Demand**
Heart rate	Basal requirement
Diastolic time	Heart rate
Coronary perfusion pressure	Wall tension
Aortic diastolic blood pressure	Preload (ventricular radius)
Ventricular end-diastolic pressure	Afterload
Arterial oxygen content	Contractility
Arterial oxygen tension	
Hemoglobin concentration	

Coronary Circulation

Right coronary artery: normally supplies the right atrium, right ventricle, and a variable portion of the posterior left ventricle.

Left coronary artery: supplies the left atrium and left ventricle (mainly anterior and lateral walls).

SA node: derived from either the right coronary artery (60%) or the left coronary artery (40%).

AV node: supplied by the right coronary artery (90%) or by the circumflex branch of the LCA (10%).

Coronary perfusion pressure (CPP): CPP = arterial diastolic pressure minus LVEDP.

Cardiac Output Determinants

- Heart Rate
  - Neural and humoral factors
- Preload
  - Venous return
    - Blood volume
    - Distribution of blood volume
      - Intrathoracic pressure
      - Posture
    - Pericardial pressure
    - Venous tone
  - Rhythm (atrial contraction)
  - Heart rate
- Afterload
  - Left ventricular radius
  - Aortic (arterial) compliance
  - Systemic vascular resistance
- Contractility
  - Neural, Humoral, and Posture
  - Pharmacological factors
  - Ventricular Compliance
- Wall Motion Abnormalities
  - Hypokinesis
  - Akinesis
  - Dyskinesis
  - Valvular Dysfunction

Mixed Venous Oxygen Saturation (SvO_2)

1. **Physiology:** SvO_2 is approximately equal to SaO_2 - (VO_2/CO x Hb).
   A. Normal range: 68-77%.
2. **Main factors** affecting SvO_2 are cardiac output, Hb, oxygen consumption (VO_2), and SaO_2.
3. **Causes of increased SvO_2**
   A. Most common cause is a permanently wedged catheter.
   B. Low VO_2 as seen with cyanide toxicity, carbon monoxide poisoning, increases in methemoglobin, sepsis, and hypothermia.
   C. High cardiac output as seen with sepsis, burns left to right shunts, atrioventricular fistulas, inotropic excess, hepatitis, and pancreatitis.
   D. High SaO_2 and high Hb are not common causes.
4. **Causes of decreased SvO_2**
   A. Decreased Hb.
   B. Increased VO_2 with fever, exercise, agitation, shivering or thyrotoxicosis.
   C. Low SaO_2 with hypoxia, respiratory distress syndrome, or inappropriate ventilatory changes.
   D. Low cardiac output as seen in myocardial infarction, CHF, or hypovolemia.
5. **Pitfalls in continuous venous oximetry**
   A. The most common errors in the continuous measurement of SvO_2 are calibration and catheter malposition. Distal migration of the PA catheter can cause an artifactually high oxygen saturation owing to highly saturated pulmonary capillary blood being analyzed.
   B. The light intensity signal may decrease if there is fibrin or deposition over the fiberoptic bundles, or if the catheter tip is lodged against a vessel wall or bifurcation.
   C. Large fluctuations in the light intensity may indicate a condition of intravascular volume deficit that allows compression or collapse of the pulmonary vasculature (especially during positive pressure ventilation).
   D. Causes of pulse oximetry artifact include the following: (1) excessive ambient light; (2) motion; (3) methylene blue dye; (4) venous pulsations in a dependent limb; (5) low perfusion; and (6) optical shunting.

Respiratory Physiology and Anesthesia

Pulmonary Function Tests

Condition	Vital Capacity*	FEV_1*	Max Vol Vent*	Re-sidual Vol*	DLCO ***	PaO_2	$PaCO_2$
Normal	>80	>75	>80	80-120	25-30	80-100	38-42
Restrictive							
Mild	60-80	>75	>80	80-120	-	nl	nl
Moderate	50-60	>75	>80	70-80	--	-	-
Severe	35-50	>75	60-80	60-70	---	-	-
Very	<35	>75	<60	<60	---	--	+
Obstructive							
Mild	>80	60-75	65-80	120-150	nl	-	nl
Moderate	>80	40-60	45-65	150-175	nl	--	-
Severe	-	<40	30-45	>200	-	--	+
Very	-	<40	<30	>200	-	---	++

*Percent predicted; **Percent vital capacity; ***ml/min/mmHg; - = Decrease; + = Increase

Pulmonary Function Tests Misc

1. **Predicted vital capacity**
   A. Women: (21.78 - [0.101 x age in years]) x height in cm.
   B. Men: (27.63 - [0.112 x age in years]) x height in cm.
2. **PFT's associated with increased pulmonary morbidity**
   A. FEV_1 < 2 liters.
   B. FEV_1/FVC < 0.5.
   C. Vital capacity < 15 ml/kg.
   D. Maximum breathing capacity < 50% of predicted.
3. **PFT's and bronchodilators**
   A. A change greater than 10-20% in FEV1 indicates reversibility.

Pediatric vs Adult Respiratory Physiology

Respiratory Parameter	Neonate	Adult
Tidal Volume (normal)	6 cc/kg	6 cc/kg
Tidal Volume (ventilated)	8-15 cc/kg	8-15 cc/kg
Dead Space	2 cc/kg	2 cc/kg
FRC	28 cc/kg	30-35 cc/kg
Oxygen Consumption	5-6 cc/kg/min	2-3 cc/kg/min
Alveolar Ventilation	100-150 cc/kg	50-60 cc/kg
VA/FRC	4.5:1	1.5:1
Closing Volume	Increased	

Normal Respiratory Parameters

Parameter	Normal Range
Tidal Vol (VT) (spont ventilation)	6 cc/kg
Tidal Volume (mech ventilation)	8-15 cc/kg
Vital Cap (VC)	60-70 cc/kg
VD/VT	33%
Min Vent	80 cc/kg

Parameter	Normal Range
FRC-adults FRC-peds	32 cc/kg 34 cc/kg
O_2 Consumption (VO_2)	3-4 cc/kg adults; 6-8 cc/kg neo/infant
Dead Space (VD)	2 cc/kg
FEV1/FVC	>75%

Hemoglobin Dissociation Curve

1. **Factors shifting the curve to the right** (decreasing hb's affinity for oxygen)
   A. Increasing hydrogen ion concentration (decreased pH).
   B. Increased 2,3-DPG concentration.
   C. Increased body temperature.
2. **P_{50}** (oxygen tension at which hemoglobin is 50% saturated)
   A. Normal adult hemoglobin: 26 mmHg.
   B. Fetal hemoglobin: 19 mmHg.
   C. Parturient maternal hemoglobin: 30 mmHg.
   D Sickle hemoglobin: 31 mmHg.
   E. Erythrocytes stored for 28 days at 1-6 °C: 17 mmHg.

Oxygenation and Ventilation

1. **Major causes of hypoxemia**
   A. Low inspired oxygen concentration (decreased FiO_2).
   B. Hypoventilation.
   C. Shunt (normal shunt about 2%): hypoxemia caused by shunt cannot be overcome by increasing the inspired oxygen concentration.
   D. Ventilation perfusion (V/Q) mismatch: common causes of V/Q mismatch are atelectasis, patient positioning, bronchial intubation, one-lung ventilation, bronchospasm, pneumonia, mucus plugging, acute respiratory distress syndrome (ARDS) and airway obstruction.
   E. Diffusion abnormalities.
   F. Cardiac output - oxygen carrying capacity abnormalties (CO/O_2 capacity): as cardiac output or oxygen carrying capacity decrease, so will oxygen delivery, which ultimatelty results in hypoxemia.

2. Major causes of hypercarbia

A. Hypoventilation: common causes include muscle paralysis, inadequate mechanical ventilation, inhalational anesthetics, and narcotics.

B. Increased CO_2 production: including malignant hyperthermia, fever, and thyrotoxicosis.

C. Iatrogenic: common examples include sodium bicarbonate admininstration and depletion of the CO_2 absorbent.

3. Methods to improve oxgenation

A. Increased FiO2.
B. Increase minute ventilation.
C. Increase cardiac output (and increase oxygen delivery to tissues).
D. Increase oxygen carrying capacity (hemoglobin).
E. Optimize V/Q relationships.
F. Cardiopulmonary bypass.
G. Decrease oxygen consumption from pain, shivering, or fever.

Arterial Blood Gases (Normal Values)

	Newborn	1-24 months	7-19 yrs	Adult	Mixed Venous	Venous
pH	7.37	7.40	7.39	7.37-7.44	7.31-7.41	7.31-7.41
PaO_2	15	90	96	80-100	35-40	30-50
$PaCO_2$	33	34	37	35-45	41-51	40-52
O_2 Sat (%)				>95	60-80	60-85
HCO_3	20	20	22	22-26	22-26	22-28

Arterial Blood Gases

1. Golden rules of ABG's

A. $PaCO_2$ change of 10 corresponds to a pH change of 0.08.
B. $PaCO_2$ increases HCO_3 conc.: initially by 1; chronically by 3.
C. $PaCO_2$ decreases HCO_3 conc.: initially by 2; chronically by 5.
D. pH change of 0.15 corresponds to BE change of 10 meq/l.

2. Total body bicarbonate deficit = base deficit (mEq/L) x pts wt (kg) x 0.4.

3. Bicarbonate deficit (HCO_3 deficit) = (total body water) x (24 - HCO_3).

4. Base excess (BE) or deficit

A. BE = HCO_3 + 10(pH - 7.40) - 24.
B. Base excess or deficit is a calculated value that gives an estimation of “acid load.”
C. Negative values of base excess (i.e., deficit) represent metabolic acidosis, and positive values indicated metabolic alkalosis.

5. During apnea, $PaCO_2$ increases 5-6 during the first minute and 3-4 for every minute thereafter.

6. Henderson-Hasselbach Equation

A. $pH = 6.1 + \log[(HCO_3)/(0.03 \times PaCO_2)]$
B. Modified equation: $(H+) = [24 \times pCO_2] / HCO_3$
C. pH = pk + log A-/HA

7. PaO2 age adjustment: $PaO_2 = 102 - (age/3)$

Saturation By PaO2

100% = 90-100	90% = 60	75% = 40	60% = 30	30% = 20
95% = 70	80% = 50	70% = 35	50% = 27	

Anion Gap

Anion gap = Na - ($Cl + HCO_3$) Normal anion gap = 8-16 mEq/l

Causes of Metabolic Acidosis

Increased Anion Gap (S.L.U.M.P.E.D.)	**Normal Anion Gap**
Salicylates	Renal causes
Lactate	Renal tubular acidosis
Uremic toxins	Carbonic anhydrase inhibitors
Methanol	GI bicarbonate loss
Paraldehyde	Diarrhea
Ethanol/ethylene glycol	Pancreatic fistula
Diabetic ketoacidosis	Ureterosigmoidostomy
	Addition of HCl
	Ammonium chloride
	Lysine or arginine HCl

Airway Innervation

Nasal Mucosa: sphenopalatine ganglion a branch of the middle division of CN V (Trigeminal Nerve). The ganglion is located on the lateral wall posterior to the middle turbinate.

Uvula, Tonsils, Superior Pharynx: innervated by continued branches from the sphenopalatine ganglion.

Oral Pharynx and Supraglottic area: innervated by branches of CN IX (Glossopharyngeal nerve). These branches include Lingual, Pharyngeal, and Tonsillar nerves.

Larynx: sensory and motor is from the Vagus (CN X).

Sensory: above the vocal folds innervated by the internal branch of the superior laryngeal nerve; below the vocal folds innervated by the recurrent laryngeal nerve.

Motor: all muscles are supplied by the recurrent laryngeal nerve except for the cricothyroid muscle which is supplied by the external branch of the superior laryngeal nerve.

Trachea: innervated by the recurrent laryngeal nerve.

Topical Nasal Agents and Anesthesia for Nasal Intubation

Local Anesthetic	Concentration	Max Dose (mg/kg)
Cocaine	4-10%	3
Tetracaine	0.5-2%	1
Hexylcaine (Cyclaine)	5%	3
Lidocaine	2-4%	3
Dyclonine (Dyclone)	0.5%	4 (for pts allergic to LA)

Vasoconstriction

- Cocaine ("Poor Man's Cocaine": 0.5-1.0% Neosynephrine + 4% lidocaine, mixed 1:1).
- Neo-Synephrine
  - Oxymetazoline (Afrin): excellent, long acting, no significant side effects.

Airway Anesthesia

1. Consider premedicating with either atropine or glycopyrrolate (to help decrease secretions) and/or sedation.
2. If considering a nasal intubation or anesthesia of the nasal airway, give 4 drops of 0.25% Neosynephrine (or afrin) to each nare to help minimize bleeding.
3. **Nerve blocks**
   - **A. Naso-tracheal nerve blocks**
     1. **Sphenopalatine ganglion (nasal mucosa)**
        - **A.** Cotton pledgets soaked with anesthetic solution (usually 20% benzocaine or 4% lidocaine) are placed in the nasal cavity at a 30 degree cephalad angulation to follow the middle turbinate back to the mucosa overlying the sphenoid bone. If you meet no resistance or if the patient gags, you are beyond the choanae and should pull back to the boney ridge of the choanae and angulate 1-2 cms superiorly.
        - **B.** A second set of pledgets is introduced through the nares and passed along the turbinates all the way to the posterior end of the nasal passage.
        - **C.** The pledgets should be left in place for at least 2-3 minutes to allow adequate diffusion of local anesthetic.
     2. **Lesser and pharyngeal palatine nerves**
        - **A. Landmarks:** 1 cm medial to the third maxillary molar and 1 cm anterior to the junction of the hard and soft palates (usually a 0.5 cm diameter spot).
        - **B.** Place a cotton pledget soaked with anesthetic solution on this site and wait 1 minute (provides topical anesthesia).
        - **C.** Using a 25 g spinal needle create a 90 degree bend 3 cm from the tip. Probe the mucosa with the needle to find the a palatine foramen (usually up to 3), angulate the needle 15 degrees medially and advance 3 cm up the canal. After negative aspiration, inject 1-3 cc of 1-2% lidocaine with epinephrine.
   - **B. Oro-tracheal nerve blocks**
     1. **Glosso-pharyngeal nerve**
        - **A.** Insert a 25 g spinal needle into the base of the posterior tonsillar pillar. After negative aspiration, inject 2-3 cc of 1-2% lidocaine with epinephrine. Repeat block on opposite side.
     2. **Superior laryngeal nerve**
        - **A**. Place the patient supine with the neck extended.
        - **B.** Find the thyrohyoid membrane (a soft depression between the hyoid and thyroid hornes) and displace the hyoid bone laterally toward the side to be blocked.
        - **C.** Insert a 25 g needle off the greater cornu of the hyoid bone inferiorly and advance 2-3 mm. As the needle passes through the thyrohyoid membrane, a slight loss of resistance is felt. Inject 2-3 cc of 1-2 % lidocaine with epinephrine. Repeat block on opposite side.

3. Translaryngeal (transtracheal)

A. Landmarks: cricothyroid membrane (located between the thyroid cartilage superiorly and the cricoid cartilage inferiorly).

B. Insert a 20 g angio-cath, bevel up, at the upper edge of cricoid cartilage in the midline. Aspirate for air to confirm placement into the trachea. Remove the needle, leaving only the angio-catheter. Inject 3-5 cc of 2-4% lidocaine solution at end inspiration. This will usually result in a vigorous cough, which aids in the spread of the solution within the trachea.

Common Indications for Tracheal Intubation

1. Provide patent airway.
2. Protection from aspiration from gastric contents.
3. Facilitate positive-pressure ventilation.
4. Operative position other than supine.
5. Operative site near or involving the upper airway.
6. Airway maintenance by mask difficult.
7. Disease involving the upper airway.
8. Protection of a healthy lung from a diseased lung (one-lung ventilation).
9. Altered level of consciousness.
10. Tracheobronchial toilet.
11. Severe pulmonary and multisystem injury associated with respiratory failure.

Complications of Endotracheal Intubation

1. Complications occurring during intubation

A. Aspiration.
B. Dental damage (chip tooth).
C. Laceration of the lips or gums.
D. Laryngeal injury.
E. Esophageal intubation.
F. Endobronchial intubation.
G. Activation of the sympathetic nervous system (high blood pressure and tachycardia).
H. Bronchospasm.

2. Complications occurring after extubation

A. Aspiration.
B. Laryngospasm.
C. Transient vocal cord incompetence.
D. Glottic or subglottic edema.
E. Pharyngitis or tracheitis.

Techniques of Intubation

1. Preparation for intubation

A. Equipment: laryngoscope with working light, endotracheal tubes of appropriate sizes, malleable stylet, oxygen supply, suction with Yankauer tip, functioning IV, etc.

1. Laryngoscope blades

A. Macintosh: the Macintosh is a curved blade whose tip is inserted into the vallecula (the space between the base of the tongue and the pharyngeal surface of the epiglottis). Most adults require a Macintosh number 3 blade.

B. Miller: the Miller is a straight blade that is passed so that the tip of the blade lies beneath the laryngeal surface of the epiglottis. The epiglottis is then lifted to expose the vocal cords. Most adults require a Miller number 3 blade.

B. Position bed height to bring the patient's head to a mid-abdominal height.

C. Head position: the correct posture of the head and neck for direct laryngoscopy is the same, regardless of the type of laryngoscope blade used or the route of intubation. Place the head in the "sniffing" position if there is no cervical spine injury or other neck pathology. The sniffing position is characterized by flexion of the cervical spine and extension of the head at the atlanto-occipital joint.

2. Orotracheal intubation

A. Position the patient's head as noted above.

B. Hold the laryngoscope in the palm of the left hand and introduce the blade into the right side of the patient's mouth. Advance the blade posteriorly and toward the midline, sweeping the tongue to the left. Check that the lower lip is not caught between the lower incisors and the laryngoscope blade.

C. When the epiglottis is in view, advance the tip of the laryngoscope blade into the vallecula if using a Macintosh blade or under the epiglottis if using a Miller blade. Check that the upper lip is not caught between the upper incisors and the laryngoscope blade.

D. Lift the laryngoscope upward and forward, in the direction of the long axis of the handle, to bring the larynx into view. Do not use the upper incisors as a fulcrum for leverage as may not only damage the upper incisors but also push the larynx out of sight.

E. The vocal cords should be visualized prior to endotracheal placement. Posteriorly, the vocal cords terminate in the arytenoid cartilages. The tube should be seen to pass between the cords, anterior to the arytenoids.

F. Pass the endotracheal tube into the pharynx with the right hand from the right side of the mouth; it should pass without resistance through the vocal cords. The endotracheal tube cuff should lie in the upper trachea but beyond the larynx.

G. Once the endotracheal tube is in place inflate the cuff, confirm endotracheal intubation (see below) and secure the endotracheal tube. In order to miminize the pressure transmitted to the tracheal mucosa, the cuff should be inflated with the least amount of air

necessary to create a seal during positive pressure ventilation. Feeling the pilot balloon is not reliable method of determining adequacy of cuff pressure. For patients intubated outside the operating room, obtain a portable chest x-ray following intubation to confirm tube placement and bilateral lung expansion.

3. Nasotracheal intubation

A. Topical cocaine (or other anesthetic method) and phenylephrine should be applied to the nasal passages.

B. Generously lubricate the nare and endotracheal tube. The endotracheal tube should be advanced through the nose directly backward toward the nasopharynx. A loss of resistance marks the entry into the oropharynx.

C. The laryngoscope and Magill forceps can be used to guide the endotracheal tube into the trachea under direct vision. For awake spontaneous breathing patients, the blind technique can be used. While listening for breath sounds at the proximal end of the endotracheal tube, advance the tube during inspiration. A cough followed by a deep breath, condensation in the tube from exhaled moisture, and loss of voice suggest tracheal entry. A fiberoptic bronchoscope can be utilized to direct the tube into the trachea.

Rapid Sequence Induction/Intubation

1. Indications: patients who are at risk for aspiration (e.g., history of recent meal, gastroesophageal reflux, pregnancy, trauma).

2. Method

A. Nonparticulate antacids, H_2-blockers or gastrokinetics may be used preoperatively to decrease the acidity and volume of gastric secretions. See premedication section.

B. Equipment is similar to that for any intubation but commonly includes several endotracheal tubes with stylet and cuff-inflation syringe in place, assortment of laryngoscope blades, functioning suction, and a patent IV.

C. Preoxygenate before induction. Four maximal breaths of 100% oxygen over 30 seconds is as effective as breathing 100% oxygen spontaneously for 3-5 minutes.

D. Induction is accomplished with any normal induction agent. Just before administration of the induction agent, cricoid pressure (Sellick's maneuver) should be applied.

E. Muscle relaxant is usually given to help facilitate intubation. Succinylcholine (1-1.5 mg/kg) is the relaxant of choice and should be given immediately after the induction agent. Once the induction agent and muscle relaxant are given, there should be no attempt to ventilate the patient by mask.

F. Intubation should be performed as soon as succinylcholine has produced jaw relaxation. Cricoid pressure should be maintained until confirmation of tracheal placement of the endotracheal tube can be made.

G. If the first attempt to intubate fails, cricoid pressure should be maintained continuously during all subsequent maneuvers, while mask ventilation with 100% oxygen is administered.

Confirmation of Tracheal Intubation

1. Direct visualization of the endotracheal tube passing though the vocal cords.
2. Carbon dioxide in exhaled gases (documentation of end-tidal CO_2 in at least three consecutive breaths).
3. Bilateral breath sounds.
4. Absence of air movement during epigastric auscultation.
5. Condensation (fogging) of water vapor in the tube during exhalation.
6. Refilling of reservoir bag during exhalation.
7. Maintenance of arterial oxygenation.
8. Chest x-ray: the tip of endotracheal tube should be between the carina and thoracic inlet or approximately at the level of the aortic notch or at the level of T_5.

Transtracheal Ventilation

1. Transtracheal ventilation can be used as a temporizing measure if mask ventilation and oxygenation become inadequate.
2. Technique: a catheter (12- or 14-gauge) is connected to a jet-type ventilator, which in turn is connected to an oxygen source capable of delivering gas at a pressure around 50 psi, and inserted into the trachea through the cricothyroid membrane. The gas is delivered intermittently by a hand-held actuator. The duration of ventilation is best assessed by watching the rise and fall of the chest: an inspiratory to expiratory ratio of 1:4 seconds is recommended.
3. Oxygenation usually improves rapidly, however, retention of carbon dioxide may limit the duration of the technique's usefulness.

Extubation Criteria

1. NIF > -20 cm H_2O
2. RR < 30/min
3. TV > 5 cc/kg
4. VC > 10 cc/kg
5. PaO_2 > 65-70 mm (FIO_2 < 40%)
6. $PaCO_2$ < 50 mm
7. Resting Min Vent < 10 l/min
8. LOC stable or improving
9. TV/RR > 10
10. Qs/Qt < 20%
11. Pmep > +40 cm H_2O
12. Vd/Vt < 0.6

Endotracheal Tube and Laryngoscope Blade Sizes

Age	**Size**
Premature	2.5
Neonate	3.0 - 3.5
6 - 12 months	3.5 - 4.0
12 - 20 months	4.0
Over 20 months	4.0 + Age (yrs.)/4
Adults (>14 years)	Female 6.5 - 8.5

Male 7.0 - 10.0

Miller 0: neonate/premature
Miller 1: up to 6 - 8 months
Wis-Hipple 1.5: 9 months- 2 yrs.
Miller 2: 2.5 - 5.0 years
Macintosh 2: child over 5 yrs.

Pediatric Airway Management (see pediatric anesthesia section)
Under 1 year: 6 + Wt.(kg)
Over 1 year: 12 + Age/2
ETT Leak: 15-20 cm H_2O

Mechanical Ventilation

1. **Classification of mechanical ventilators**
   - **A. Time cycled:** the tidal volume is delivered and inspiration ends after a preset time interval.
   - **B. Volume cycled:** the tidal volume is delivered and inspiration ends after a preset time interval.
   - **C. Pressure cycled:** the tidal volume is delivered and inspiration ends when a preset volume is delivered.
2. **Modes of mechanical ventilation**
   - **A. Intermittent positive-pressure ventilatory modes (IPPV)**
     1. **Controlled mechanical ventilation (CMV):** mechanical breaths are delivered at a preset rate and tidal volume regardless of the patient's effort.
     2. **Assist-control ventilation (AC):** a preset minute ventilation is delivered regardless of the patient's effort. The ventilator senses each patient-initiated spontaneous breath and delivers a preset tidal volume as well.
     3. **Intermittent mandatory ventilation (IMV):** the ventilator provides tidal volume breaths at a preset fixed rate. In between ventilator-delivered breaths, the patient is able to breathe spontaneously at any rate, tidal volume, or pattern.
     4. **Synchronized intermittent mandatory ventilation (SIMV):** similar to IMV, with the ventilatory breaths timed to coincide with spontaneous effort.
     5. **Continuous positive airway pressure (CPAP):** a preset level of positive airway pressure is maintained throughout the respiratory cycle. The patient must be spontaneously breathing.
     6. **Inspiratory pressure support ventilation (IPS):** a preset pressure is obtained when the patient initiates an inspiratory effort (pressure-cycle ventilation).
     7. **Pressure-controlled Ventilation**
        - A. Maximum airway pressure is set on the ventilator, and tidal volume becomes the dependent variable.
        - B. The duration of inspiration is determined by setting either the inspiratory time or the I:E ratio. Tidal volume is the product of inspiratory flow and inspiratory time.
        - C. The primary advantage of pressure-controlled ventilation is reduction in peak airway pressure and potential improvement of gas exchange.
     8. **High frequency ventilation**
        - A. High frequency positive pressure ventilation (HFPPV): similar to conventional ventilation, however, tidal volumes are very small, and cycling frequencies are very fast (60-300).
        - B. High frequency jet ventilation (HFJV): a small diameter injecting catheter positioned in the central airway pulses gas along the luminal axis under high pressure at a rapid cycling rate.
     9. **Pressure-controlled inverse ratio ventilation (PC-IRV):** IRV is set by choosing a prolonged inspiratory time such that the time spend during inspiration exceeds expiratory time.

10. **Airway pressure release ventilation (APRV):** allows the spontaneously breathing patient to make ventilatory efforts around an elevated pressure baseline (CPAP) but allows the system to depressurized (partially or completely) for brief periods.

3. PEEP (positive end-expiratory pressure)

A. Function of PEEP

1. PEEP increases oxygenation by maximizing the ventilation-perfusion relationship in the lung. PEEP does this by maximizing the FRC (functional residual capacity), keeping lung volumes greater than closing capacity, therefore maintaining airways open and functional.

B. Adverse effects of PEEP

1. Decreased cardiac output.
2. Hypotension.
3. Worsening hypoxia.
4. Barotrauma (pneumothorax).
5. Increased intracranial pressure.
6. Decreased urine output.

4. Ventilator Settings

A. FIO_2: normally start with 40% otherwise use 90-100% until first ABG available (1% decrease in FIO_2 = decrease PaO_2 by 7).

B. PEEP: initially none; start with 5 cm H_2O and increase in 3-5 cm H_2O increments if PaO_2 less than 60 mmHg with FIO_2 > 50%; over 10 cm H_2O normally requires pulmonary artery catheter.

C. Rate: start at 10-14 (for infants start at 25-30).

D. Tidal volume: 10-15 ml/kg (infants 8-12 ml/kg).

E. Mode: IMV, SIMV, CPAP, A/C, PSV.

Oxygen Therapy

1. Nasal cannulas: FIO_2 increases by 3-4% per liter of oxygen given (up 40-50%).

2. Masks

A. Simple mask: simple mask may deliver oxygen flow rates from 6-15 liters per minute providing FIO_2 of 0.35-0.65.

B. Venturi mask(air entrainment mask): delivers up to 50% FIO_2 with accuracy.

C. Partial rebreathing mask: simple mask with a valveless reservoir bag and exhalation ports. Can deliver up to 80% FIO_2.

D. Nonrebreathing mask: simple mask with reservoir bag and unidirectional valve. Can deliver up to 95% FIO_2.

E. Aerosol face tent: delivers oxygen form variable oxygen nebulizer over mouth and nose.

Laryngeal Mask Airway (LMA)

1. Indications for LMA

A. In place of a face mask or endotracheal tube in a spontaneously breathing anesthetized patient.

B. In place of an endotracheal tube, when the breathing is being controlled, as long as the inflation pressure is not more than 20 cm H_20.

C. To aid in the management of the difficult airway (i.e., the LMA can be

used as a guide for fiberoptic intubation).

2. Contraindications for LMA

A. The LMA does not provide an airtight seal of the airway and, thus, does not protect against gastric regurgitation and pulmonary aspiration.

B. When controlled ventilation is likely to require a high-inflation pressure of more than 20 cm H_2O.

3. Insertion of the LMA

A. Propofol (2.5-3.0 mg/kg) is the agent of choice for LMA insertion. Propofol tends to relax the jaw and pharyngeal muscles better than thiopental.

B. The leading edge of the deflated cuff should be wrinkle-free and facing away from the aperture. Lubricate only the back side of the cuff with a water soluble lubricant.

C. The LMA is held like a pencil and is inserted via the mouth blindly in the midline with concavity forward, while pressing on the anterior shaft with the tip of the index finger toward the hard palate and guiding it toward the pharynx.

D. When the upper esophageal sphincter is reached, a characteristic resistance is felt. The cuff is then inflated with air (normally the cuff should be inflated without holding the tube to enable the expanding cuff to find its correct position in the pharynx).

E. When correctly placed, the black vertical line on the posterior aspect of the tube should always face directly backward, toward the head of the patient.

F. The LMA should be left in place until the patient can open their mouth on command. During emergence, the patient should not be stimulated (i.e., suctioned), and the cuff should not be deflated (until the patient can open his/her mouth on command).

4. Complications

A. Possibility of regurgitation of gastric contents and pulmonary aspiration.

B. Oral and pharyngeal mucosa injury during insertion of the LMA.

C. Laryngospasm and coughing (may occur if the LMA is inserted in a lightly anesthetized patient).

D. Negative pressure pulmonary edema after improper placement in spontaneously breathing patient

5. Cleaning and sterilization

A. The LMA is reusable and must be steam sterilized before each use.

B. Clean with a mild soap or dilute sodium bicarbonate solution. Do not expose valve to any cleaning agent.

C. Deflate cuff completely prior to autoclaving.

D. Do no use chemicals including Cidex or ethylene oxide to sterilize.

6. LMA sizes (ETT= endotracheal tube; * = cuffed tube)

Size	Patient	Cuff Vol (ml)	Largest ETT
1	Infant up to 6.5 kg	4	3.5
2	Infants/Children up to 20 kg	10	4.5
2.5	Children between 20 - 30 kg	15	5.0
3	Children/small adults over 30 kg	20	6.0*
4	Average adults	30	6.0*
5	Large adults greater than 80 kg	30	7.0*

Esophageal Tracheal Combitube (ETC)

1. **Uses:** emergency airway control in the difficult airway. Available only in one adult size (age > 15 years and height > 5 feet)
2. **Insertion**
   - A. With the head in the neutral position, insert the ETC, with gentile pressure, up to the black marks (teeth should be between black marks).
   - B. Inflate the first pilot balloon (blue cuff) with 100 cc. As the cuff is inflate the combitube will pop out some (approximately 1 cm) similar to the LMA.
   - C. Inflate the second pilot balloon (white cuff) with 10-15 cc.
3. **Placement**
   - A. Ventilate via longer (blue) lumen.
   - B. If breath sounds are present, the ETC is in the esophagus; continue to ventilate.
   - C. If no breath sounds are heard, change ventilation to shorter lumen #2 (clear) and recheck for breath sounds. If breath sounds are present, the ETC is in the trachea; continue to ventilate.
   - D. If no breath sounds or breath sounds faint, attempt to improve seal by adding up to 60 cc to balloon number 1.
   - E. If unable to ventilate, deflate both cuffs, pull back 3 cm and reinflate cuffs. Ventilate via blue lumen and check for breath sounds. If still no breath sounds, deflate cuffs, remove ETC and start algorithm over.
4. **Contraindications**
   - A. Height less than 5 feet (only one size currently available).
   - B. Intact gag reflex intact (will not tolerate cuff).
   - C. Presence of esophageal disease (potential for bleeding or rupture).
   - D. Ingestion of caustic substances (potential for rupture).
   - E. Upper airway obstruction (foreign body, glottic edema, epiglottis).
5. **Concerns**
   - A. Potential for nasopharyngeal, oropharyngeal or tracheal mucosal damage or edema (particularly if left in for greater than 2-8 hours).
   - B. Inability to suction tracheal secretions when in the esophageal position.
   - C. Only one size available; single use makes it expensive.

Bullard Laryngoscope

1. The Bullard laryngoscope, functioning as an indirect fiberoptic laryngoscope, provides direct visualization of the vocal cords. It is available in both adult and pediatric sizes.
2. The advantage of this laryngoscope is that it can be introduced into the oropharynx with minimal mouth opening (oral opening of 0.64 cm required) and the patient can remain in anatomical position.
3. Preloading the intubating stylet involves lubricating the stylet and positioning the endotracheal tube so that the distal end of the stylet projects through the Murphys eye of the endotracheal tube.
4. The blade is then inserted into the mouth, with the handle in the horizontal plane, and rotated into the vertical plane allowing it to slide around the midline of the tongue and into the posterior pharynx. Gentle traction is applied against the posterior surface of the tongue to obtain visualization of the glottic aperture. With the stylet pointed directly at the glottic opening, the endotracheal tube is advanced under direct vision into the trachea. As with other retraction blades, a considerable amount of practice is required to become skilled with its use.

Breathing Systems

1. Open systems

A. Open systems have neither a reservoir bag nor permit rebreathing (i.e. the anesthetic agents are delivered directly to the patient). In these systems fresh gas supply consists of atmospheric air and there is no significant apparatus dead space.

B. Types of open systems

1. Insufflation: insufflation is delivery of gas directly across the patient's face.
2. Open drop administration: open drop administration is achieved by dripping the anesthetic liquid onto layers of gauze stretched over a wire frame in the shape of a face mask. This method of administration is rarely used today.

C. Advantages of open systems

1. Simple and inexpensive.
2. Little or no resistance to breathing.

D. Disadvantages of open systems

1. Unstable anesthetic state and wasteful of anesthetic agents.
2. No reservoir bag to assess respiration (unable to assist or control ventilation).

2. Semiopen anesthetic breathing systems

A. Components of Mapleson circuits

1. Breathing tubes: made out of corrugated breathing tubes.
2. Fresh gas inlet.
3. Pressure relief valve (overflow valve).
4. Breathing bag (reservoir bag).
5. There are no valves or carbon dioxide absorbers.

B. Mapleson circuits

1. Mapleson A system

A. Characterized by placement of the overflow valve near the patient and separation of the gas reservoir bag from this valve by a corrugated tube. No one-way valves.

B. The Mapleson A system is the most efficient Mapleson circuit for spontaneous ventilation.

2. Mapleson B system

A. The expiratory valve and fresh gas supply are close to the patient and the reservoir bag is placed distally, separated by corrugated tubing.

3. Mapleson C system

A. Overflow valve, fresh gas flow, and reservoir bag are all close to the patient. Essentially the same as Mapleson B except that there is no corrugated tubing to separate fresh gas from mixed gas in the distally place reservoir bag.

4. Mapleson D system

A. Fresh gas supply is close to the patient, reservoir bag and expiratory valve are placed distally separated by corrugated tubing.

B. The Mapleson D system is the most efficient Mapleson circuit for controlled ventilation.

C. Bain System

1. Bain system is a coaxial version of Mapleson D system in which the fresh gas flow enters through a narrow tube within a corrugated expiratory limb.
2. The primary advantages of a Bain system include, warming of inspiratory gas by the surrounding exhaled gas, improved humidification by partial rebreathing, and ease of scavenging exhaled gases.
3. Primary disadvantages include, high fresh gas flow requirements, accidental kinking or disconnection of the inner tube may lead to dangerous level of rebreathing and hypercapnia, and increased resistance to breathing.

5. Mapleson E system

A. Similar to a Mapleson D system without a reservoir bag or expiratory valve. Also called a Ayre's T-Piece.

B. Controlled ventilation not possible without occluding corrugated tubing (there is no bag on this system).

6. Mapleson F system

A. Similar to a Mapleson E system with the addition of a reservoir bag at the end with an opening to the exhalation limb such that controlled ventilation can be achieved. Also called a Jackson-Rees.

7. Advantages of semi-open Mapleson systems

A. Simplicity: easy to assemble and use.
B. Light weight.
C. Portable.
D. Low resistance.

8. Disadvantages of semi-open Mapleson systems

A. High fresh gas flow requirements results in waste of anesthetic gases; loss of heat and humidity.
B. Gas flow settings difficult and confusing.
C. Potential for excessive rebreathing and hypercapnia.

D. Scavenging difficult.

9. Misc info on the Mapleson systems

A. The most efficient Mapleson system for spontaneous breathing: Mapleson A > Mapleson D > Mapleson C > Mapleson B (ADCB: **A**ny **D**ummy **C**an **B**reathe).

B. The most efficient Mapleson system for controlled ventilation: Mapleson D > Mapleson B > Mapleson C > Mapleson A (DBCA).

3. Semi-closed systems

A. To and fro system.

B. Circle system (most widely used anesthetic system)

1. Components of the circle system

A. Carbon dioxide absorbent: most commonly soda lime or barium hydroxide lime. Carbon dioxide chemically combines with water to form carbonic acid which is neutralized by the carbon dioxide absorbents. Color conversion of a pH indicator dye by increasing hydrogen ion concentration signal absorbent exhaustion.

B. Unidirectional valves: one-way valves (located between the patient and the reservoir bag on both the inspiratory and expiratory limbs of the circuit) ensure that exhaled gas is not rebreathed without passing through the carbon dioxide absorber.

C. Reservoir bag with an adjustable pressure limiting valve located on the expiratory limb.

D. Fresh gas inflow.

2. Advantages of circle system

A. Low fresh gas flows required.

B. Easy scavenging, resulting in lower operating room and environmental pollution.

C. Conserves heat and humidity.

D. Spontaneous and controlled ventilation are both easily achieved.

E. Relatively small dead space.

3. Disadvantages of circle system

A. Increased resistance to breathing.

B. Greater size and complexity (increased potential for malfunction).

C. Potential for hypoxic gas mixture and decreased inspired concentration of soluble anesthetics due to low fresh gas flows.

D. Difficultly with cleaning and sterilizing.

Laboratory Values

CSF

Glucose	40-70 mg/dL
Total Protein	20-45 mg/dl
CSF Pressure	50-180 mm H_2O
Leukocytes	Total<4 per mm3
Lymph	60-70%
Mono	30-50%
Neutro	1-3%

Renal

Cr Clearance	
Males	125 ml/min
Females	105 ml/min
Ur Creat	1.0-1.6 g/d
Ur Protein	<0.15 g/d
Ur K	25-100 meg/d
Ur Na	100-260 meg/d

Chemical

Acid Phosphatase	0-5.5 U/L
Albumin	3.5-5.5 g/dL
Alk Phosphatase	30-120 U/L
Aminotransferases	
AST (SGOT)	0-35 U/L
ALT (SGPT)	0-35 U/L
Ammonia	80-110 mcg/dL
Amylase	60-80 U/L
Bilirubin	
Total	0.3-1.0 mg/dL
Direct	0.1-0.3 mg/dL
Indirect	0.2-0.7 mg/dL
Calcium	8.6-10.5 mg/dL
CO_2	22-30 meq/L
Chloride	98-106 meq/L
Cholesterol	
Total (years)	
<29	<200 mg/dL
30-39	<225 mg/dL
40-49	<245 mg/dL
>50	<265 mg/dL
HDL	30-90 mg/dL
LDL	50-190 mg/dL

CPK	25-145 U/L
Creatinine	0.4-1.5 mg/dL
Ferritin	15-200 ng/ml
Glucose	70-140 mg/dL
Iron	80-180 mcg/dL
Iron-Binding	250-450 mcg/dL
Iron-Sat	20-45
LDH	25-100 U/L
Lipase	49-220 U/L
Magnesium	1.6-2.6 mg/dL
Osmolality	285-295
Phosphorus	2.5-4.5 mg/dL
Protein	5.5-8.0 meq/L
Sodium	136-145 meq/L
Triglycerides	<60 mg/dL
Urea nitro	10-20 mg/dL
Uric Acid	
Males	2.5-8.0 mg/dL
Females	1.5-6.0 mg/dL

Laboratory 104

Complete Blood Count

Male	1 month	6-12 years	Adult
WBC	5.0-19.5	5.0-13.5	4.5-11.0
RBC	3.0-5.4	4.0-5.2	4.6-6.2
Hb	14.0-18.0	11.5-15.5	14.0-18.0
Hct	31-55	35-45	42-52
RDW			11.5-14.5

Female	1 month	6-12 years	Adult
WBC	5.0-19.5	5.0-13.5	4.5-11.0
RBC	3.0-5.4	4.0-5.2	4.2-5.4
Hb	14.0-18.0	11.5-15.5	12.0-16.0
Hct	31-55	35-45	37-47
RDW			11.5-14.5

Leukocytes

Total 4500-11000 cells/uL

Differential (Approx % of Total)

Segs	40-70%	Lymphs	20-50%	Baso	0-3%
Bands	1-5%	Mono	2-6%	Eosino	0-5%

ESR Males: 0-15 mm/h Females: 0-20 mm/h

Fibrinogen 150-360 mg/dl

Miscellaneous Lab Information

1. **Calculated osmolality** = 2 Na + glucose/18 + BUN/2.8 + ethanol/4.6 + isopropanol/6 + methanol/3.2 + ethylene glycol/6.2 (norm 280-295).

Electrolyte Disturbances

1. **Calcium**
   A. Normal plasma concentration is 8.5-10.5 mg/dl with 50% free ionized 40% protein bound.
   B. Normal free ionized concentration is 4.5-5 mg/dl.
   C. Corrected calcium = measured calcium / [0.6 + (total protein / 8.5)].
   D. For each 1 gm/dl change in albumin there is a corresponding 0.8 mg/dl change in total calcium (free ionized calcium is not affected by albumin changes).
   E. Ionized calcium increases 0.16 mg/dl for each decrease of 0.1 unit in plasma pH.
2. **Glucose**
   A. For each 100 mg/dl glucose above normal there is a corresponding fall in sodium by 1.6 meq/l.

Fluid and Electrolyte Management

1. **Functional fluid compartments**
   - **A. Total body water (TBW):** 60% (adult males) or 50% (adult females) of ideal body weight (IBW).
   - **B. Intracellular fluid (ICF):** comprises approximately 35% of IBW or 60% of TBW. Principal potassium containing space.
   - **C. Extracellular fluid (ECF):** accounts for 25% of IBW or 40% of TBW and is subdivided into interstitial fluid (ISF) and blood volume (BV; approximately 8% of TBW). Principal sodium containing space.
2. **Guidelines for intraoperative crystalloid fluid replacement**

Insensible losses	2 ml/kg/hr
Minor trauma/surgery	3-4 ml/kg/hr
Moderate trauma/surgery	5-6 ml/kg/hr
Major trauma/surgery	7-8 ml/kg/hr

3. **Maintenance fluid requirements**

First 10 kg	4 ml/kg/hr
Second 10 kg	2 ml/kg/hr
>20 kg	1 ml/kg/hr

4. **Daily electrolyte requirements**
   - Na: 2-3 mEq/kg/24 hours
   - K: 1-2 mEq/kg/24 hours
   - Cl: 2-3 mEq/kg/24 hours
5. **Determinants of perioperative fluid requirements**
   - A. Basal requirements.
   - B. Preoperative deficits.
   - C. Third-space losses.
   - D. Transcellular fluid losses.
   - E. Effects of anesthetic agents and technique.

IV Fluids

Fluid	glu gm/L	Na	Cl	K	Ca	HCO_3	Kcal/L
D_5W	50						170
0.5 NS		77	77				
NS		154	154				
D5 .25NS	50	38	38				170
LR		130	110	4	3	27	<10

Blood Therapy Management

1. **Blood loss management**
   - **A. Estimated blood volume (EBV) equals**
     1. 100-120 ml/kg for premature infant
     2. 90 ml/kg for full-term infant
     3. 80 ml/kg for infants 3 to 12 months
     4. 70 ml/kg thereafter
   - **B.** Replace every 1 ml blood loss with 3 ml crystalloid or 1 cc pRBC
   - **C.** pRBC: one unit pRBC increases Hct about 3% and Hb about 1 g/dl
     1. 3 ml/kg pRBC increases Hb about 1 g/dl
     2. 10 ml/kg pRBC increases Hct about 10%
   - **D. Max allowable blood loss** (MABL) = [EBV x (starting Hct - target Hct)] / starting Hct
   - **E. Fluid replacement equivalents**
     1. Crystalloid: 3 cc/1 cc estimated blood loss [EBL]
     2. Colloid: 1 cc/cc EBL
     3. Whole Blood: 1 cc/cc EBL
     4. pRBC: 1/2 cc/cc EBL
2. **Compatibility testing**
   - **A. Type specific**: ABO-Rh typing only; 99.80% compatible.
   - **B. Type and screen:** ABO-Rh and screen; 99.94% compatible.
   - **C. Type and crossmatch:** ABO-Rh, screen, and crossmatch; 99.95% compatible.
   - **D. Screening donor blood:** hematocrit is determined, if normal, the blood is typed, screened for antibodies, and tested for hepatitis B, hepatitis C, syphilis, HIV-1, HIV-2, and human T-cell lymphotropic viurses I and II. ALT is also commonly measured as a surrogate marker of nonspecific liver infection.
3. **Blood component therapy**
   - **A. Whole blood**: 40% hct; used primarily in hemorrhagic shock.
   - **B. Packed red blood cells (pRBC):** 70% hct; each unit contains 250-300 ml volume and rises Hb approximately 1 g/dl.
   - **C. Platelets:** single donor bag (10-25 ml/bag) or multiple donor bag (50-70 ml/bag); approximately 5.5 x 10^5 platelets per unit; each unit increases platelet count 5000-10000; platelets are the only blood product stored at room temperature.
   - **D. Fresh frozen plasma (FFP):** 250 cc/bag; contains 200 units of procoagulants and plasma proteins including factors V and VIII.
   - **E. Cryoprecipitate:** 10-20 ml/bag; contains 100 units factor VIII-C, 100 units factor vWF, 60 units factor XIII, and 250 mg fibrinogen; used for factor VIII deficiency and Hemophilia A.
   - **F. Albumisol:** 5% and 25% (heat treated at 60 degrees C for 10 hrs).
4. **Blood storage solutions**
   - **A.** Federal regulation determines the duration of blood storage and requires that at least 70% of transfused red blood cells survive 24 hours after transfusion. Storage times depend on the preservative solution.
   - **B. CPD:** citrate prevents clotting by binding calcium, phosphate acts as a buffer, and dextrose allows cells to continue glycolysis and maintain sufficient ATP; 21-day storage limit.

C. CPD-A1: similar to CPD, adds adenine which increases survival by allowing RBC to resynthesize ATP and extra dextrose for prolonged storage; 35-day storage limit.

D. Adsol: a 100 ml volume of Adsol contains 2.2 gms of dextrose, 27 mg of adenine, 750 mg of mannitol, and 900 mg of NaCl, and when added to a unit of CPD preserved RBC's storage limit is increased to 42 days.

5. Complications

A. Immune/non-hemolytic

1. **Febrile:** most common (0.5-4.0% of transfusions); due to recipient antibodies against donor WBC's and platelets; treat with slow infusion and antipyretics.
2. **Allergic:** occurs in about 3% of transfusions; caused by immunoglobulin alloantibodies against substances in the donor plasma with activation of mast cells and histamine release; most commonly present with abrupt onset of pruritic erythema or urticaria on arms and trunk; treat with slow infusion and antihistamines.
3. **Anaphylaxis:** occurs in IgA deficient patients who have developed an anti-IgA; immune complex activates mast cells, basophils, etc. resulting in hypotension, dyspnea, laryngeal edema, wheezing and possibly shock; treat like severe allergic reaction.
4. **Acute lung injury:** form of noncardiogenic pulmonary edema; donor antibody combines with recipient leukocytes which leads to trapping of aggregates in the lungs and the release of lysosomal contents.

B. Immune/hemolytic

1. **Acute hemolytic transfusion reaction:** occurs 1:10,000 with 20-60% mortality; usually due to donor blood ABO incompatibility; complement activation leads to hemolysis and may result in DIC; clinically presents with HA, chills, N/V, fever, flank pain, hypotension, dyspnea, bleeding and hemoglobinuria; ARF may occur from stromal and lipid precipitation in tubules.
2. **Delayed hemolytic transfusion reaction:** occurs 1:2500; usually seen in previously sensitized patients (prior transfusions, multiparous); results in destruction by RES in 2-21 days.

C. Non-immune

1. **Infections**
   - **A. AIDS:** overall risk about 1:225,000 per unit.
   - **B. Hepatitis B:** risk is currently 1:200,000 per unit; accounts for 2% of transfusion related hepatitis.
   - **C. Hepatitis C:** risk is currently 1:3300 per unit; accounts for 98% of transfusion related hepatitis.
   - **D. HTLV 1 and 2:** 4% chance of developing T-cell leukemia or spastic paraparesis; risk is 1:50,000 per unit.
2. **Metabolic**
   - **A. Decreased pH:** reflecting lactate production.
   - **B. Increase potassium:** due to cell lysis; increases with length of storage.

C. **Decrease in 2,3 DPG:** consumed by RBC's; P_{50} decreases to 18 mmHg after 1 week and 15 mmHg after 3 weeks.
D. **Decreased plasma bicarbonate.**
E. **Increased PCO2.**

3. **Coagulopathy**
   A. **Usually occurs only after massive transfusion** (greater than 10 units).
   B. **Dilutional thrombocytopenia:** most common cause of abnormal bleeding in massive transfusion, responds quickly to platelet transfusions.
   C. **Low Factors V and VIII:** factors V and VII are very labile in stored blood and may decrease to levels as low as 15-20% normal, however, this is usually enough for hemostasis.
   D. **DIC:** DIC is a hypercoagulable state caused by activation of the clotting system leading to deposition of fibrin in microvasculature which causes a secondary activation of fibrinolysis: results in consumption of factors and platelets.
4. **Miscellanous**
   A. **Citrate toxicity**: citrate binds calcium which leads to hypocalcemia (rarely occurs unless transfusion given faster then 150 cc/70 kg/min).
   B. **Hypothermia**: small decreases in temperature can lead to post-op shivering and increase oxygen consumption; at 30°C ventricular irritability and cardiac arrest can occur.
   C. **Microaggregates:** white cell aggregates take at least 5 days to form; cellular debris may not be filtered by microfilter (no significant decrease in the incidence of ARDS with micropore filter).

6. Massive transfusions

A. General information

1. Massive transfusion is arbitrarily definied as the replacement of a patient's total blood volume in less than 24 hours, or as the acute administration of more than half the patient's estimated blood volume per hour.
2. The use of universal donor blood (group O, Rh negative blood)
   A. Group O, Rh negative blood should be reserved for patients close to exsanguination. If time permits, cross-matched or uncross-matched type specific blood should be administered.
   B. Group O, Rh negative blood should not be given as whole blood. The serum contains high anti-A and anti-B titers that may cause hemolysis of recipient red cells.
   C. If more than 4 units of group O, Rh negative <u>whole blood</u> is administered, type-specific blood should not be given subsequently since the potentially high anti-A and anti-B titers could cause hemolysis of the donor blood.
   D. Patients administered as much as 10 units of group O, Rh

negative packed red blood cells may be switched to type-specific blood, since there is an insignificant risk of hemolysis from the small volume of plasma administered with packed red blood cells.

B. Complications of massive transfusions

1. **Hypothermia:** every effort should be made to use blood warmers. All blood products should be given through the blood warmer except platelets and cryoprecipitate
2. **Impaired oxygen release** from hemoglobin: erythrocytic levels of 2,3-DPG decrease with stored blood (oxygen dissociation curve shifts to the left).
3. **Coagulopathy**
   A. Dilutional thrombocytopenia is the most frequent bleeding disorder seen in trauma patients, but rarely becomes a problem until at least 150-200% of the patient's blood volume has been replaced. However, thrombocytopenia can occur following smaller transfusions if disseminated intravascular coagulation (DIC) occurs or there is pre-existing thrombocytopenia.
   B. Platelet activity in stored blood is only 5-10% of normal after 24-48 hours of storage.
   C. Most coagulation factors are stable in stored blood except factors V and VIII.
   D. Platelets should not be given through the level one or rapidly infused.
4. **Electrolytes and acid base abnormalities**
   A. Hyperkalemia or hypokalemia can occur with massive blood transfusion.
   B. The plasma potassium concentration of stored blood increases during storage and may be over 30mmol/l. Hyperkalaemia is generally not a problem unless very large amounts of blood are given quickly. On the contrary, hypokalaemia is more common as red cells begin active metabolism and intracellular uptake of potassium restarts.
   C. Acid-base status should be followed with blood gases. Lactic acid levels in the blood pack give stored blood an acid load of up to 30-40mmol/l. This, along with citric acid is usually metabolised to bicarbonate, and a profound metabolic alkalosis may ensue.
5. **Citrate toxicity**
   A. Citrate intoxication is due to acutely decreased serum ionized calcium, because citrate chelates calcium.
   B. Most believe empiric administration of calcium is not warranted unless ionized calcium is low.
   C. Each unit of blood contains approximately 3g citrate, which binds ionized calcium. The healthy adult liver will metabolise 3g of citrate every 5 minutes. Transfusion at rates higher than one unit every five minutes or impaired liver function may thus lead to citrate toxicity and hypocalcaemia.

6. Microaggregates

A. Microaggregates begin forming after 2 days of blood storage.

B. No conclusive evidence that micropore filters are beneficial.

7. Infection

A. HIV: 1:250,00 donor unit exposures.

B. Post-transfusion hepatitis constitutes the principal viral risk of blood transfusion (incidence of 4-15%).

7. Alternatives to homologous blood transfusion

A. Autologous transfusion.

B. Donor-directed transfusion.

C. Perioperative blood salvage (cell saver).

D. Intraoperative isovolemic hemodilution.

E. Use of substitute products for replacement of plasma and blood volume.

Hemolytic Transfusion Reaction

1. Treatment

A. STOP THE TRANSFUSION.

B. Maintain the urine output at a minimum of 75 to 100 ml/hr by the following methods

1. Generously administer fluids IV and possibly mannitol, 12.5 to 50 grams, given over a 5-15 minute period.
2. If IV administered fluids and mannitol are ineffective, than administer furosemide, 20-40 mg IV.

C. Alkalinize the urine since bicarbonate is preferentially excreted in the urine, only 40-70 mEq/70 kg of sodium bicarbonate is usually required to raise the urine pH to 8, whereupon repeat urine pH determinations indicate the need for additional bicarbonate.

D. Assay urine and plasma hemoglobin concentrations.

E. Determine platelet count, PTT, serum fibrinogen level.

F. Return unused blood to blood bank for crossmatch and send blood sample for antibody screen and direct antiglobulin test.

G. Prevent hypotension to ensure adequate renal blood flow.

Spinal and Epidural Anesthesia

1. **Contraindications to peridural anesthesia**
    - **A. Absolute contraindications**
        1. Lack of patient consent.
        2. Localized infection at injection site.
        3. Generalized sepsis or bacteremia.
        4. Allergy to local anesthetics.
        5. Increased intracranial pressure.
        6. Coagulopathy.
    - **B. Relative contraindications**
        1. Localized infection peripheral to regional site.
        2. Demyelinating central nervous system disease.
        3. Chronic back pain or prior lumbar spine surgery.
        4. Hypovolemia.
        5. Patients taking platelet inhibiting drugs.
2. **Spinal anesthesia** (local anesthetic placed in the subarachnoid space)
    - **A. Anatomy**
        1. **Spinal canal:** extends from the foramen magnum to the sacral hiatus.
        2. **Spinal cord:** spinal cord extends the length of the vertebral canal during fetal life, ends at L3 at birth, and moves progressively cephalad to reach the adult position of L1-L2 by 2 years of age.
        3. **Subarachnoid space:** subarachnoid space lies between the pia mater and the arachnoid and extends from S2 to the cerebral ventricles.
        4. **Course of anatomy to the subarachnoid space:** skin, subcutaneous tissue, supraspinous ligament, interspinous ligament, ligamentum flavum, epidural space, and dura.
    - **B. Physiological changes with spinal and epidural anesthesia**
        1. **Neural blockade**
            - A. Sequence of neural blockade
                1. Sympathetic block with peripheral vasodilation and skin temperature elevation.
                2. Loss of pain and temperature sensation.
                3. Loss of proprioception.
                4. Loss of touch and pressure sensation.
                5. Motor paralysis.
            - B. The above sequence of neural blockade occurs because smaller C fibers are blocked more easily than the larger sensory fiber, which in turn are blocked more easily than motor fibers. As a result, the level of autonomic blockade extends above the level of the sensory blockade by 2-3 segments, while the motor blockade is 2-3 segments below the sensory blockade.
            - C. With epidural anesthesia, the local anesthetics act directly on the spinal nerve roots located in the lateral part of the space. As a result, the onset of the block is slower than with spinal anesthesia, and the intensity of the sensory and motor block is less.

2. **Cardiovascular**
   A. Hypotension: the degree of hypotension is directly proportional to the degree of sympathetic blockade.
   B. Blockade above T4 interrupts cardiac sympathetic fibers leading to bradycardia, decreased cardiac output, and further decrease in blood pressure.
3. **Respiratory**
   A. With ascending height of the block into the thoracic area, there is a progressive, ascending intercostal muscle paralysis. Tidal volume, minute ventilation, and maximum inspiratory volume are all maintained by the diaphragm. The diaphragmatic ventilation is mediated by the phrenic nerve, and typically will remain unaffected even during high cervical blockade.
4. **Visceral effects**
   A. Bladder: sacral blockade results in an atonic bladder.
   B. Intestine: with sympathectomy, vagal tone dominates and results in a small, contracted gut with active peristalsis.
5. **Renal**
   A. Renal blood flow and GFR are unaffected by central block due to autoregulation, except with extreme hypotension.
6. **Neuroendocrine**
   A. Central blockade can block the complex hormonal and metabolic stress response to surgery.
7. **Thermoregulation**
   A. Vasodilation that results from the sympathetic blockade predisposes patients to hypothermia.

3. Factors influencing spinal anesthetic

- **A. Dosage.**
- **B. Drug volume** (greater the volume the greater the spread within CSF).
- **C. Addition of vasoconstrictors** (decrease vascular uptake and prolong action).
- **D. Baricity of the local anesthetic** solution (specific gravity).
- **E. Shape of the spinal canal** (when supine, a high point at L3-L4 and low point at T5-T6).
- **F. Position of the patient.**
- **G. Intra-abdominal pressure** (increased intra-abdominal pressure, as seen with pregnancy, obesity, ascites, or abdominal tumors, increases the blood flow through the epidural venous plexus, reducing the volume of CSF, thus causing the local anesthetic to spread further.
- **H. Age** (spinal space thought to become smaller with age).

4. Complications of spinal anesthesia

- **A. Hypotension:** prehydrating with 500-1000 cc of crystalloid before performing the block will help decrease the incidence of hypotension.
- **B. Paresthesia or nerve injury:** during placement of the needle or injection of anesthetic, direct trauma to a spinal nerve or intraneural injection may occur.

C. **Blood tap or vascular injury:** needle may puncture an epidural vein during needle insertion.
D. **Nausea and vomiting:** usually the result of hypotension or unopposed vagal stimulation.
E. **High spinal:** may see apnea with total spinal from direct blockade of C3-C5.
F. **Pain on injection.**
G. **Backache:** overall the incidence of backache following spinal anesthesia is no different from that following general anesthesia.
H. **Postdural puncture headache:** usually seen 6-48 hours after dural puncture (see below).
I. **Urinary retention:** urinary retention may outlast the sensory and motor blockade.
J. **Infection:** meningitis, arachnoiditis, and epidural abscess may occur, but are exceedingly rare.

5. **Factors influencing epidural anesthesia**
   A. **Local anesthetic selected.**
   B. **Mass of drug injected** (dose, volume, and concentration).
   C. **Addition of vasoconstrictors** (epinephrine) reduces systemic absorption.
   D. **Site but not speed of injection** or patient position.
   E. **Patients over 40 years of age.**
   F. **Pregnancy** (hormonal and/or mechanical factors).
6. **Complications of epidural anesthesia**
   A. **Dural puncture:** unintentional dural puncture occurs in 1% of epidural injections performed.
   B. **Catheter complications**
      1. Inability to insert the catheter.
      2. Catheter can be inserted into an epidural vein.
      3. Catheters can break off or become knotted within the epidural space.
   C. **Unintentional subarachnoid injection.**
   D. **Intravascular injection:** may result in local anesthetic overdose where relatively large amounts of local anesthetic are used.
   E. **Direct spinal cord injury:** possible if the injection is above L2 in the adult patient.
   F. **Bloody tap:** may result from perforation of an epidural vein.
7. **Postdural puncture headache**
   A. **Characteristics of a postdural puncture headache**
      1. **Postural component** (made worse by upright position).
      2. **Frontal or occipital location.**
      3. **Tinnitus.**
      4. **Diplopia.**
      5. **Young females.**
      6. **Use of a large-gauge needle.**
   B. **Mechanism**
      1. Usually due to a continued leak of CSF through the hole in the dura mater, resulting in low CSF pressure, which causes traction on meningeal vessels and nerves.
      2. Incidence: the overall incidence is approximately 5-10%.

C. Treatment of a postdural puncture headache

1. **Oral Analgesics.**
2. **Bed rest.**
3. **Hydration** (IVF, PO fluids, and caffeine containing beverages).
4. **Caffeine infusion** (500 mg caffeine and sodium benzoate in 1 liter of isotonic crystalloid given over 1-2 hours).
5. **Epidural blood patch** (placement of 10-20 cc of autologous blood, especially if HA persists >24 hours or is severe). The success rate with an epidural blood patch is approximately 95%.

Neuraxial Opioids: side effects and treatment

Problem	Treatment Options	Notes
Pruritus	Nalbuphine 5-10 mg IV/IM Diphenhydramine 25-50 mg IV Naloxone 40-80 mcg IV	May be severe after intrathecal morphine
Nausea/ Vomiting	Metoclopramide 5-10 mg IV Nalbuphine 5-10 mg IV/IM Naloxone 40-80 mcg IV	
Respiratory Depression	Naloxone 0.1 mg IV prn	Uncommon with fentanyl or sufentanil Watch for synergism with other sedatives
Urinary Retention	Urinary Catheter	
Blood Pressure Changes	Fluid hydration Ephedrine Phenylephrine	Most likely after meperidine (local anesthetic effects)

Regional Anesthesia

Regional Anesthesia

1. General information

A. Consent for general anesthesia should always be obtained even when only a peripheral nerve block is planned.

B. Regardless of which type of nerve block is planned, standard ASA monitors (blood pressure, pulse oximetry, EKG, etc) should be used.

C. General information on techniques for peripheral nerve blocks

1. Paresthesia
   A. Placing a needle in direct contact with a nerve or within the substance of the nerve will stimulate that nerve causing a parasthesia.
   B. Injection into a perineural location often results in a brief accentuation of the parasthesia; in contrast, an intraneural injection produces an intense, searing pain that signals the need to immediately terminate the injection.
2. Correct needle placement can be determined by elicitation of parasthesia (see above), perivascualr sheath technique, transarterial placement, and a nerve stimulator.

D. Regional block needles

1. Blunt-bevel needle: designed to minimize trauma upon direct contact with nerves. The angle of the bevel is increased 20-30 degrees, and the sharpness is decreased.
2. Insulated needle: a nonconductor is bonded to the needle except for the last millimeter before the bevel.
3. The beaded needle: a regional needle designed for use with a nerve stimulator.

2. Brachial plexus block

A. Interscalene block

1. Technique: the needle is inserted in the interscalene groove at the level of the cricoid cartilage and advanced perpendicular to the skin in all planes until a paresthesia is elicited or a transverse spinous process is cotacted, at which point 30-40 cc of local anesthetic is injected.
2. Indications: any procedure on the upper extremity, including the shoulder. The high rate of failure to achieve full block of the ulnar nerve (10-20%) must be kept in mind when performing this technique for hand surgery.
3. Special contraindications: contralateral phrenic paresis, severe asthma.
4. Side effects: Horner's syndrome, phrenic paresis.
5. Complications: proximity of the vertebral artery makes intra-arterial injection possible with rapid progression to grand mal seizure after small amounts are injected. The neural foramina can be reached, and massive epidural, subarachoid, or subdural injection can occur. Stellate ganglion block results in Horner's sign (myosis, ptosis, anhidrosis). Other complications include recurrent laryngeal

nerve block (30-50%) leading to hoarseness, phrenic nerve block, pneumothorax, infection, bleeding, and nerve injury.

B. Supraclavicular block

1. Indications: operations on the upper arm, elbow, lower arm and hand.
2. Special contraindications: hemorrhagic diathesis, contralateral phrenic paresis.
3. Side effects: Horner's syndrome, phrenic paresis.
4. Complications: penumothorax (1-6%) and hemothorax are the most common. Phrenic nerve block and Horner's syndrome may occur.

C. Axillary block

1. Indications: operations on the lower arm and hand.
2. Anatomy: it should be noted that in the axilla, the musculocutaneous nerve has already left its sheath and lies within the coracobrachialis.
2. Special contraindications: lymphangitis (presumed infected axillary nodes).
3. Complications: puncture of the axillary artery, intravenous/intra-arterial injection (systemic toxic reaction), postoperative neuropathies (more common when multiple sites of paresthesia are elicited).

2. Techniques to identify placement of the needle

A. Fascial "clicks": feel a "click" as the needle advances and penetrates the sheath.

B. Paresthesia technique: patient cooperation is essential. Instruct the patient to say "stop" if and when they experience

Pediatric Anesthesia

Basic Pediatric Anesthesia Drugs

Atracurium	0.5 mg/kg	STP (IV)	4-6 mg/kg
Atropine	0.01-0.02 mg/kg	STP (PR)	25-30 mg/kg
Fentanyl	3-5 mcg/kg	Succinylcholine	1-1.5 mg/kg
Glycopyrrolate	5-10 mcg /kg	Vecuronium	0.1 mg/kg
Neostigmine	0.05 mg/kg	Versed (IV)	0.07-0.08 mg/kg

Miscellaneous Pediatric Anesthesia Drugs

Chloral Hydrate	50-100 mg/kg	Methohexital (PR)	25-30 mg/kg
Diazepam (IV)	0.1 mg/kg	Methohexital (IV)	1-2 mg/kg
Droperidol	0.01-0.05 mg/kg	Morphine	0.05-0.1 mg/kg
Ephedrine	0.1 mg/kg	Naloxone	0.1 mg/kg
Furosemide	0.2-1 mg/kg	Pentobarbital (IM)	4-6 mg/kg
Ketamine (IV)	1-2 mg/kg	Phenylephrine	1-2 mcg/kg
Ketamine (IM)	5-10 mg/kg	Tylenol	10-15 mg/kg
Meperidine (IV)	0.02-2.0 mg/kg	Versed (nasal)	0.3 mg/kg
Meperidine (IM)	1 mg/kg	Versed (PO)	0.5-1.0 mg/kg

Pediatric Emergency Drugs

Epinephrine	0.01-0.02 mg/kg	Epinephrine	0.1-1.0 mcg/kg/min
Verapamil	0.1-0.3 mg/kg	Dopamine	2-20 mcg/kg/min
Na Bicarbonate	0.5-2 mEq/kg	Dobutamine	5-20 mcg/kg/min
peds 8.4%:	1 mEq/cc	Isoproterenol	0.1-1.0 mcg/kg/min
neonatal 4.2%:	0.5 mEq/cc	Nitroprusside	0.5-8 mcg/kg/min
Calcium Cl	10-30 mg/kg	Lidocaine	20-50 mcg/kg/min
Ca Gluconate	30-60 mg/kg	Norepi	0.1-1 mcg/kg/min
Lidocaine	1 mg/kg	Digoxin	0.02-0.04 mg/kg
Bretylium	5-10 mg/kg	Defibrillation	2-4 J/kg
Glucose (D25%)	0.5-1.0 gm/kg	Cardioversion	0.25-0.5 J/kg

Pediatric Airway Management

Pediatric Endotracheal Tube Sizes and Laryngoscope Blade Size

Age	**Size**
Under 1500 gm	2.5
1500-5000 gm	3.0
Infant	3.0 - 3.5
6 - 12 months	3.5 - 4.0
12 - 20 months	4.0
Over 20 months	4.0 + Age (yrs)/4

Miller 0: neonate/premature
Miller 1: up to 6 - 8 months
Wis-Hipple 1.5: 9 months- 2 yrs
Miller 2: 2.5 - 5.0 years
Macintosh 2: child over 5 yrs

Pediatric Endotracheal Tube Recommendations

1. Endotracheal Tube Leak: 15-20 cm H_2O.
2. Length of Insertion of ETT
   A. Under 1 year: 6 + Wt(kg).
   B. Over 1 year: 12 + Age/2.
3. Uncuffed ETT generally used for patients under 10 yrs.

Pediatric Vital Signs

Age	RR	HR	SBP	DBP
Preterm	60	140	50	
Neonate	40	140	65	40
12 months	30	120	95	65
3 years	25	100	100	70
12 years	20	80	110	60

Age And Approximate Weight

28 weeks = 1 kg +/- 100 g/wk from 22-30 wks
<1 year: 1/2 age (months) plus 4 kg
1 yr to puberty: 2 times age in yrs plus 10 kg

Physiologic Differences (as compared to an adult)

1. **Cardiac:** cardiac output of neonates and infants is dependent on heart rate, since stroke volume is relatively fixed by a noncompliant and poorly developed left ventricular. The sympathetic nervous system and baroreceptor reflexes are not fully mature. The hallmark of hypovolemia is hypotension without tachycardia.
2. **Respiratory:** increased respiratory rate; tidal volume and dead space per kg constant; lower functional residual capacity; lower lung compliance; greater chest wall compliance. Alveolar maturation is not complete until late childhood, and small alveoli are associated with low lung compliance. Hypoxic and hypercapnic ventilatory drives are not well developed in neonates and infants.
3. **Metabolism/temperature:** higher ratio of body surface area to body weight; heat production in neonates is nonshivering thermogenesis by metabolism of brown fat.
4. **Renal:** normal renal function by 6 months of age; premature neonates may posses decreased creatinine clearance, impaired sodium retention, glucose excretion, and bicarbonate reabsorption. Hypoglycemia is defined as < 30 mg/dL in the neonate, and <40 mg/dL in older children.

Anatomical Differences

1. **Airway:** larger head and tongue, narrow nasal passages, cephalad (and anterior appearing) larynx (opposite vertebra C4 versus C6 in adults), long epiglottis, short trachea and neck; cricoid cartilage narrowest point of airway in children younger than 5 years of age (glottis in adults).

Pharmacologic Differences

1. **Pediatric drug dosing** is commonly based upon a per-kilogram recommendation. A child's weight can be roughly estimated, based upon age (see above).
2. **Inhalational anesthetics**: higher alveolar ventilation, relatively low functional residual capacity (ie, a higher ratio of minute ventilation to functional residual capacity), and a large vessel-rich group contribute to a rapid rise in alveolar anesthetic concentration. Furthermore, the blood/gas coefficients of isoflurane and halothane are lower in neonates than adults. The minimum alveolar concentration is higher in infants than in neonates or adults. The blood pressure of neonates and infants tends to be more sensitive to volatile anesthetics.

3. **Nonvolatile anesthetics:** some barbiturates and opioid agonist appear to be more toxic in neonates than adults, possibly secondary to easier entry across the blood-brain barrier, decreased metabolic capability, or increased sesitivity of the repiratory centers.
4. **Muscle relaxants:** infants require higher doses of succinylcholine per kilogram than do adults because of their larger volume of distribution. Children are more subject to cardiac dysrhythmias, myoglobinemia, hyperkalemia, and malignant hyperthermia after succinylcholine than adults. The response of neonates to nondepolarizing muscle relaxants is quite variable. Immaturity of the neuromuscular junction (particularly in premature neonates) tends to increase sensitivity.

Anesthetic Considerations for Specific Pediatric Disorders

1. **Prematurity:** prematurity is defined as birth before 37 weeks gestation or weight less than 2500 grams; premature infants are at increased risk for retinopathy of prematurity; premature infants less than 50 (some say 60) weeks postconception are prone to episodes of apnea up to 24 hours postoperatively (should be monitored for 12-24 hours postoperatively). Risk factors for postanesthetic apnea include anemia, hypothermia, sepsis, and neurologic abnormalities.
2. **Congenital diaphragmatic hernia:** three types (left or right posterolateral foramen of Bochdalek or anterior foramen of Morgagni) with left most common; a reduction in alveoli and bronchioli (pulmonary hypolpasia) is accompanied by marked elevation in pulmonary vascular resistance; hallmarks include hypoxia, scaphoid abdomen, bowel in the thorax; gastric distention should be minimized by placement of a nasogastric tube and avoidance of high levels of positive-pressure ventilation; sudden fall in lung compliance, blood pressure or oxygenation may signal a contralateral pneumothorax.
3. **Tracheoesophageal fistula:** most common is combination of upper esophagus that ends in a blind pouch and a lower esophagus that connects to the trachea; breathing results in gastric distention; intubated awake without muscle relaxants. Aspiration pneumonia and the coexistence of other congenital anomalies are common. The key to successful mangement is correct endotracheal tube position (the tip of the tube should lie between the fistula and the carina).
4. **Hypertrophic pyloric stenosis:** persistent vomiting depletes sodium, potassium, chloride, and hydrogen ions, causing hypochloremic metabolic alkalosis. Initially, the kidney tries to compensate for the alkalosis by excreting sodium bicarbonate in the urine. Later, as hyponatemia and dehydration worsen, the kidneys must conserve sodium even at the expense of hydrogen ion excretion (resulting in paradoxic aciduria). Correction should be with sodium chloride supplemented with potassium. Electrolyte abnormalities must be corrected prior to surgery. These neonates may be at increased risk for respiratory depression and hypoventilation in the recovery room because of persistent metabolic or cerebrospinal fluid alkalosis.
5. **Gastroschisis**: defect in abdominal wall (lateral to umbilicus); no hernial sac; no associated congenital anomalies. The neonate remains intubated after the procedure and is weaned from the ventilator over the next 1-2 days.

6. **Omphalocele:** defect in abdominal wall at the base of the umbilicus; hernial sac present; associated with congenital anomalies (trisomy 21, cardiac anomalies, diaphragmatic hernia, bladder anomalies).
7. **Infectious croup:** obstruction of the airway characterized by a barking cough; usually follows upper respiratory infection (parainfluenza); age 3 months to 3 years; progresses slowly; rarely requires intubation.
8. **Acute epiglottis:** bacterial infection (Hemophilus influenza type B); most commonly in 2 to 6 year olds; progresses rapidly from a sore throat to dysphagia and complete airway obstruction; drooling; treat with antibiotics and intubation (laryngoscopy should not be performed before induction of anesthesia because of possibility of laryngospasm).

Additional Special Problems in Pediatric Anesthesia

1. **Acute porphyria**: Extreme sensitivity to barbiturates.
2. **Asthma:** Bronchospasm with curare; adrenal insufficiency after withdrawal of corticosteroids.
3. **Cystic fibrosis:** Impacted bronchial secretions with positive airway pressure; post-operative hypercapnia, hypoxemia.
4. **Extensive burns/trauma:** Sudden severe hyperkalemia and cardiac arrest after succinylcholine.
5. **Familial dysautonomia (Riley-Day Syndrome):** Circulatory instability with hypertension or hypotension and vomiting during anesthesia; hyperpyrexia; recurrent bronchopneumonia.
6. **Glycogen storage disease:** Metabolic acidosis and hypoglycemia during anesthesia.
7. **Hurler, Pierre-Robin, Treacher-Collins Syndromes:** Severe upper airway obstruction when unconscious; difficult tracheal intubation.
8. **Leukemia and malignancies:** Cardiotoxicity if treated with Adriamycin (more than 450 mg/m^2).
9. **Myasthenia Gravis:** Prolonged neuromuscular blockade after non-depolarizing relaxants; resistant to depolarizing relaxants.
10. **Myotonias:** Severe, intractable muscle spasm after succinylcholine; malignant hyperthermia.
11. **Neuroblastoma/Pheochromocytoma:** Severe hypertension with tumor manipulation requiring adrenergic blockade.
12. **Renal Tubular Disease:** Prolonged neuromuscular blockade after gallamine due to delay excretion.
13. **Reyes Syndrome:** Elevated intracranial pressure.
14. **Sickle Cell Anemia:** Sickling crisis with decreased PaO_2, pH, thromboses (CNS, renal) with dehydration.

Anesthesia for Cardiac Surgery

Classification of Congenital Heart Defects

1. **Left to right shunt** (increased pulmonary blood flow)
   - A. Atrial septal defect (ASD).
   - B. Ventricular septal defect (VSD).
   - C. Patent ductus arteriosus.
2. **Right to left shunt (decrease pulmonary blood flow)**
   - A. Tetralogy of Fallot (RVH, overriding aorta, VSD, pulmonic stenosis).
   - B. Pulmonary atresia.
   - C. Ebstein's anomaly.
3. **Mixing of systemic and pulmonary circulations**
   - A. Truncus arteriosus.
   - B. Transposition of the great vessels.
   - C. Total anomalous pulmonary venous drainage.
4. **Obstructive lesions**
   - A. Aortic stenosis.
   - B. Pulmonary stenosis.
   - C. Coarctation of the aorta.

Pediatric Cardiovascular Physiology

1. **Fetal circulation**
   - A. Acidosis, sepsis, hypothermia, hypoxia, and hypercarbia may cause reopening of the fetal shunts and persistence of the fetal circulation.
   - B. Diagnosis of persistent pulmonary hypertension of the newborn can be confirmed by measurement of the PaO_2 in blood obtained simultaneously from preductal (right radial) and postductal (umbilical, posterior tibial, dorsalis pedis) arteries. A difference of 20 mmHg verifies the diagnosis.
2. **Closure of the ductus arteriosus**
   - A. In the fetus, patency of the ductus arteriosus is maintained by high levels of prostaglandin (PGI_2 and PGE_1).
   - B. Functional closure occurs by contraction of the smooth muscle of the ductal wall and usually occurs 10-15 hours after birth. An increase in PO_2 and a decrease in prostaglandins at birth contribute to functional closure.
   - C. Permanent anatomic closure of the duct usually occurs in 4 to 6 weeks.
3. **Closure of the foramen ovale**
   - A. Increase in left atrial over right atrial pressure functionally closes the foramen ovale.
   - B. Anatomic closure of the foramen ovale occurs between 3 months and 1 year of age, although 20%-30% of adults and 50% of children less than 5 years of age have a probe-patent foramen ovale.
4. **Closure of the ductus venosus**
   - A. Decrease in umbilical venous blood flow causes passive closure of the ductus venosus.
   - B. The ductus venosus is functionally closed by 1 week of life and anatomically closed by 3 weeks.

Valvular Heart Disease

Disease	Heart Rate	Rhythm	Preload	Afterload	Contractility	Blood Pressure
Aortic Stenosis	normal (70-80) avoid tachy	sinus is essential	increase or maintain	maintain don't decrease	maintain	maintain
Aortic Regurg-itation	normal or slight increase	sinus	maintain or increase	reduce	maintain or increase	maintain
Mitral Stenosis	normall (70-80) avoid tachy	sinus, A-fib ok, should digitalize	maintain or increase	maintain, avoid increased PVR	maintain	avoid hypo-tension
Mitral Regurg-itation	normal or increase avoid brady	usually A-fib, should digitalize	maintain	reduce	maintain or increase	maintain
Ischemic Heart Dz	slow rate	sinus	maintain	reduce	maintain or decrease	normal at rest
IHSS	normal or slight decrease	sinus, consider pacing	maintain or increase	maintain or slight increase	maintain or decrease	maintain

Premedications for Cardiac Surgery

A. Premedication for adult patients

1. Common premedications for cardiac surgery include: morphine 0.1-0.15 mg/kg IM, scopolamine 0.3-0.4 mg IM, and plus/minus diazepam 0.15 mg/kg or lorazepam 0.06 mg/kg PO approximately 1-2 hours prior to surgery. The dose of scopolamine, however, should be reduced to 0.2 mg for patients older than 70 years of age.
2. The dose of premedication should be reduced or possibly avoided in patients with critical aortic or mitral stenosis, those undergoing cardiac transplantation, patients with CHF, and patients with renal or hepatic dysfunction.
3. For patients on heparin, they should not receive any IM medications. A common predication for heparinized patients includes diazepam 0.15 mg/kg PO (or lorazepam 0.04 mg/kg) and morphine 1-10 mg IV.
4. Premedications should be given approximately 1-2 hours prior to the patient coming down to the operating room.

B. Premedications for pediatric patients

1. IM premedication

A. Under 6 months: atropine 10-20 mcg/kg IM (minimum 100 mcg).
B. 6-12 months: atropine 10-20 mcg/kg IM (minimum 100 mcg) plus morphine 0.10-0.15 mg/kg.

C. Over one year: atropine 10-20 mcg/kg IM (minimum 100 mcg) or scopolamine 0.015 mg/kg IM (maximum of 0.4 mg) or glycopyrrolate 0.004 mg/kg IM (maximum of 0.3 mg) plus morphine 0.1-0.2 mg/kg IM.

2. Oral premedication

A. Under 6 months: atropine 20 mcg/kg PO.
B. 6-12 months: pentobarbital 2-4 mg/kg PO +/- PO atropine.
C. Over one year: pentobarbital 2-4 mg/kg PO + Demerol 1-2 mg/kg PO (0.5-1.0 mg/kg demerol in one year olds) +/- PO atropine.

C. Other orders and medications

1. Current cardiac medications should be continued; diuretics are usually held except in patients with CHF or afternoon cases.
2. Nasal canal oxygen (2-4 liters per minute) should be ordered along with the premedication order (i.e., oxygen should be given to all patients given premedications).
3. Patients undergoing rigid bronchoscopy or therapeutic bronchoscopy should receive an anticholinergic to minimize secretions (glycopyrrolate 0.2 mg IM).
4. Document on the anesthesia record what premedications were ordered.

Intraoperative Management for Cardiac Surgery

A. The mnemonic **LAMPS** (l, laboratory values; A, anesthetics and anesthesia machine; M, monitors; P, patient/pump; S, support) has been used by some to aid in organizing appropriate information relating to cardiopulmonary bypass.

B. Checklist prior to initiating cardiopulmonary bypass

1. ACT or measure of adequate heparinization (ACT >400 seconds ideal).
2. Nitrous oxide off (if used).
3. Pulmonary artery catheter should be pulled back 3-5 cm.
4. Turn transesophageal echo off.

C. Checklist during cardiopulmonary bypass

1. ACT or measure of adequate heparinization.
2. Frequent ABG's (uncorrected), hematocrit, potassium, calcium levels.
3. Discontinue ventilation after the heart stops ejecting and you are on cardiopulmonary bypass at full flow.
4. Watch for
   A. Hypotension
      1. Venous cannula: kink, malposition, clamp, air lock.
      2. Inadequate venous return: bleeding hypovolemia, IVC obstruction.
      3. Pump: poor occlusion, low flows.
      4. Arterial cannula: misdirected, kinked, partially clamped, dissection.
      5. Vasodilation: anesthetics, hemodilution, idiopathic.
      6. Transducer or monitoring malfunction, stopcocks the wrong way.
   B. Hypertension
      1. Pump: increased flow.
      2. Arterial cannula: misdirected.
      3. Vasoconstriction: light anesthesia, response to

temperature changes.
4. Transducer or monitor malfunction.
5. Monitor patients' facial appearance: suffusion (inadequate SVC drainage), unilateral blanching (innominate artery cannulation).
6. Check adequacy of perfusion by monitoring flow and pressure, acidosis, and mixed venous oxygen saturation.

D. Checklist prior to terminating cardiopulmonary bypass

1. Check labs: hematocrit (ideal to be 22-25%), ABG's, potassium, and calcium.
2. Lungs ventilated with 100% oxygen.
3. Look at the heart to evaluate overall function.
4. Core temperature should be at least 37°C.
5. Stable rhythm (preferably sinus rhythm) with adequate heart rate (80-100 beats/min).
6. All monitors on and recalibrated.

Acid-Base Management During CPB

A. pH-stat: requires temperature correction for interpretation of blood gases during CPB. Temperature correction can be accomplished by setting the blood gas analyzer to measure the sample the patient's temperature. To achieve a pH of 7.40 and a $PaCO_2$ of 40 mmHg when the patient is cold requires the addition of CO_2.

B. alpha-stat: requires no temperature correction for interpreting blood gases. The sample is warmed to 37 degrees C. and then measured in the blood gas analyzer as any other sample. The addition of CO_2 is usually not necessary.

Post-Cardiopulmonary Bypass Bleeding

A. Differential diagnosis

1. Uncorrected surgical defects.
2. Circulating anticoagulants: residual heparin and heparin rebound, protamine anticoagulation
3. Platelet defects: platelet function defect (most common cause of post-CBP bleeding after heparin is reversed and surgical bleeding is controlled), thrombocytopenia.

B. Treatment

1. Circulating anticoagulants: adequate heparinization should be confirmed with ACT, and additional protamine given if needed.
2. Platelet abnormalities: usually given after other coagulation deficiencies have been corrected and no surgically correctable lesion exists (irrespective of actual number of circulating platelets).
3. Deficiencies of circulating procoagulants: corrected by infusing FFP, cryoprecipitate, or fresh donor blood.

C. Prevention

1. **Pharmacological factors**
   - **A. Desmopressin**
     1. Synthetic product that increases plasma levels of Factor VIII and Von Willebrand factor and decreases bleeding times.
     2. Dosing: 0.3 mcg/kg IV given over 20-30 min.

3. Side effects: decreased free water clearance from ADH activity, hypotension, thrombosis, decreased serum sodium, hyponatremic seizures.

B. Aprotinin

1. Inhibitor of several proteases and factor XIIa activation of complement (see selected drug section).

C. Antifibrinolytic agents

1. Epsilon aminocaproic acid (Amicar)

A. Synthetic antifibrinolytic; inhibits proteolytic activity of plasmin and conversion of plasminogen to plasmin by plasminogen activator.

B. Dose: Loading dose 100-150 mg/kg IV followed by constant infusion of 10-15 mg/kg/h.

2. Transexamic acid

A. Similar mechanism as epsilon aminocaproic acid but is approximately 10 times more potent.

B. Dose: loading dose 10 mg/kg IV followed by constant infusion of 1 mg/kg/h.

3. Complications of antifibrinolytics

A. Bleeding into kidneys or ureters may thrombose and obstruct the upper urinary tract.

B. Contraindicated if DIC.

C. Hypotension may occur with rapid administration.

D. May be associated with thrombosis and subsequent stroke, myocardial infarction or deep vein thrombosis.

Automatic Implantable Cardioverter Defibrillator (AICD)

A. Common indications for AICD implantation

1. Patients with a history of near-sudden death who have not responded to drug therapy and are not candidates for arrhythmia surgery.
2. Patients who have had unsuccessful arrhythmia surgery.
3. Post cardiac arrest patients who have not had an MI and who have no inducible arrhythmia during electrophysiologic testing.
4. Patients undergoing endocardial resection for recurrent VT.

B. Contraindications

1. Uncontrolled congestive heart failure.
2. Frequent recurrences of VT such that the AICD battery would rapidly deplete.

C. Surgical techniques

1. Nonthoracotomy approach: this approach employs either a single or multiple endocardial electrodes positioned fluoroscopically.
2. Thoracotomy approaches
   A. Median sternotomy.
   B. Left thoracotomy.
   C. Subxiphoid approach.
   D. Subcostal approach.

D. Intraoperative testing

1. The purpose of intraoperative testing is to establish the defibrillation threshold (i.e., the minimum energy required to defibrillate the heart to a stable rhythm). Internal paddles should be readily available in the operative field during the entire procedure, and external

patches should also be placed preoperatively.

2. The initial step in testing the system is to ensure adequate sensing capability.
3. The leads are connected to an external cardioverter-defibrillator unit. This device can deliver programmable shocks of 1 to 40 J to defibrillate the heart.
4. Ventricular fibrillation is induced via rapid ventricular pacing or alternating current. A period of 10 to 20 seconds of fibrillation follows in order to ensure that spontaneous cardioversion will not occur.
5. The ECD unit is activated and discharged to determine the defibrillation threshold (the lowest amount of energy required to reliably attain a stable rhythm. Approximately 6 to 8 defibrillations are usually necessary to test the system. Amiodarone or hypokalemia may be responsible for high defibrillation thresholds.
6. If all the tests performed with the ECD unit are successful, an AICD is connected to the leads and ventricular fibrillation is again induced to test the newly implanted unit.

E. Anesthetic considerations

1. Preoperative assessment

A. A thorough preoperative evaluation should be preformed. The indication for the AICD should be noted.

B. Many patients will be taking antidysrhythmic agents at the time of surgery. In theory, the device and defibrillation thresholds should be tested while the patient is on the drug regimen that is planned postoperatively.

C. Antidysrhythmic agent of concern is amiodarone, which is negative inotropic agent and vasodilator. Amiodarone may cause refractory bradycardia or may precipitate a profound and prolonged hypotensive state postoperatively.

2. Intraoperatively monitoring: standard monitors and an arterial line are the minimum required monitors. Central venous access may be considered for administration of vasoactive drugs. Pulmonary catheter is depends on the patients cardiac status. Usually, however, a pulmonary artery catheter is not required for this procedure.

3. Anesthetic technique: general anesthesia with nitrous oxide, narcotic, and muscle relaxant anesthetic is most common. This technique has minimal effects upon the induction of VT.

4. Other considerations

A. The cardioversion is commonly associated with transient hypertension and tachycardia, probably caused by sympathetic outflow.

B. Multiple intraoperative inductions of VT or VF may cause profound hypotension.

C. External defibrillator must be available.

D. Isoproterenol infusion occasionally has been administered to facilitate dysrhythmia induction.

E. The AICD is occasionally inactivated after being placed to avoid the cautery from trigger the AICD to discharge. The AICD is reactivated postoperatively.

5. **AICD complications**
   A. Pacemaker interaction
      1. Both temporary and permanent pacemakers may interact with a AICD by interfering with dysrhythmia detection.
      2. The AICD can be deactivated when using temporary pacing, especially, A-V sequential pacing.
   B. Mechanical: lead fractures, lead insulation breaks, and lead migration.
   C. Rate miscounting leading to unnecessary shocks.
   D. Infection.

Anesthesia for Elective Cardioversion

A. Preoperative evaluation

1. The patient should be evaluated as receiving a general anesthetic in the operating room. Patients should NPO prior to the procedure.
2. 12 lead electrocardiogram should be preformed before the procedure to confirm that the arrhythmia is still present.
3. Preoperative labs should be within normal limits (metabolic and electrolyte disorders should be corrected).

B. Monitoring

1. Standard ASA monitors should be used, as for any general anesthetic.
2. Reliable intravenous access.
3. Other equipment should include ambu bag, suction, airway supplies, anesthetic drugs, and crash cart.

C. Anesthetic technique

1. Premedication is not usually necessary.
2. Brief period of amnesia or a light general anesthetic is all that is usually required. This can be accomplished with etomidate, methohexital, propofol, or a benzodiazepine.
3. Following preoxygenation with 100% oxygen, the sedative-hypnotic is given. As soon as consciousness is lost, the appropriate charge may be delivered. The airway is maintained and ventilation is supported until consciousness is regained.

D. Complications of cardioversion

1. Transient myocardial depression, postshock arrhythmias, and arterial embolism.

Vascular Surgery

1. **Anesthesia for carotid artery surgery** (carotid endarterectomy; CEA)
   - **A. Preoperative considerations**
     1. Indications: TIAs associated with ipsilateral severe carotid stenosis (>70% occlusion), severe ipsilateral stenosis in a patient with a minor (incomplete) stroke, and 30-70% occlusion in a patient with ipsilateral symptoms (usually an ulcerated plaque), emboli arising from a carotid lesion, amaurosis fugax, large ulcerated plaque (symptomatic but without high grade stenosis), surgical team with low stroke/death rate.
     2. Operative mortality is 1-4% and is primarily due to cardiac complications.
     3. Perioperative morbidity is 4-10%. Stroke is the most common and expected major complication during and after carotid endarterectomy. The incidence of stroke varies with the indication for surgery. Hypertension occurs in about 70% of patients undergoing carotid endarterectomy and is associated with an increase in the risk of stroke since cerebral blood flow may be reduced and autoregulation impaired.
     4. Complications of carotid endarterectomy: hematoma with tracheal compression, supraglottic edema, cranial nerve injury (cranial nerves VII, IX, X, and XII), myocardial infarction, intraparenchymal hemorrhage, carotid occlusion, intracerebral hemorrhage, embolism.
   - **B. Preoperative anesthetic evaluation**
     1. Most patients undergoing CEA are elderly and hypertensive, with generalized arteriosclerosis. Preoperative evaluation should include a through cardiac and neurologic evaluation.
   - **C. Anesthesia technique**
     1. The anesthesia goal is to maintain adequate cerebral perfusion without stressing the heart. In addition, the patient should be sufficiently responsive immediately after surgery to obey commands and thereby facilitate neurologic evaluation.
     2. Regardless of the anesthetic agents selected, mean arterial blood pressure should be maintained at or slightly above the patient's usual range. During carotid occlusion blood pressure should be maintained at or up to 20% higher than the patient's highest recorded resting blood pressure while awake.
     3. Blood pressure and heart rate changes during and after carotid surgery are quite variable. Surgical manipulation of the carotid sinus can cause an increase in afferent impulses to the brainstem and trigger an abrupt bradycardia and hypotension. This may be prevented by infiltration of the sinus with local anesthetic. If infiltration has not been performed, then clamp application may cause hypertension and tachycardia since the sinus is now sensing a low pressure. The reverse may or may not be observed with

unclamping. The high degree of variability between individuals in this reflex behavior may be due to differing degrees of sinus insensitivity secondary to the atherosclerotic process.

4. Ventilation should be adjusted to maintain normocapnia. Hypocapnia can produces cerebral vasoconstriction, while hypercapnia can induce intracerebral steal phenomenon. Thus most recommend the maintenance of normocarbia during carotid endarterectomy.
5. Heparin (5000-10,000 units IV) is usually given prior to occlusion of the carotid artery. Protamine, 50-75 mg, can be given for reversal prior to skin closure.

D. Monitoring

1. Intraarterial blood pressure monitoring is mandatory.
2. Additional hemodynamic monitoring should be based primarily on the patients underlying cardiac function. However, it should be noted that carotid endarterectomy is not usually associated with significant blood loss or fluid shifts.
3. Cerebral monitoring
   A. Electroencephalogram (EEG) and somatosensory evoked potentials (SSEP) have been used to determine the need for a shunt. Electrophysiologic signs of ischemia after cross-clamping can dictate the use of a shunt. Although multichannel recordings and computer processing can enhance the sensitivity of the EEG, neither EEG nor SSEP monitoring is sufficiently sensitive or specific to reliably predict the need for shunting or the occurrence of postoperative deficits. In fact no study to date has demonstrated that the use of these methods improves outcome.
   B. Other monitoring methods include: measurements of regional cerebral blood flow with radioactive xenon 133, transcranial doppler measurement of middle cerebral artery flow velocity, carotid stump pressure distal to the cross-clamp (ideal greater the 60 mmHg), jugular venous oxygen saturation, and transconjuctival oxygen tension. These methods, however, are not reliable.

E. Regional anesthesia

1. Regional anesthesia is advocated by some and can be achieved by performing a superficial and deep cervical plexus block, which effectively blocks the C2-C4 nerves.
2. The principal advantage of this technique is that the patient remains awake and can be examined intraoperatively: thus, the need for a temporary shunt can be assessed and any new neurologic deficits diagnosed during surgery.
3. Disadvantages of regional anesthesia include patient discomfort and loss of cooperation, confusion, panic, or seizures. The awkwardness of these possibilities discourages the majority from using the technique.

F. Postoperative considerations

1. Postoperative hypertension may be related to surgical denervation of the ipsilateral carotid baroreceptor.

Hypertension can stress and rupture the surgical anastomosis resulting in the development of a wound hematoma, which can rapidly compromise the airway.

2. Transient postoperative hoarseness and ipsilateral deviation of the tongue may occur. They are due to surgical retraction of the recurrent laryngeal and hypoglossal nerves, respectively.

2. Anesthesia for surgery of the aorta

A. Ascending aorta

1. Surgery routinely uses median sternotomy and cardiopulmonary bypass.
2. Anesthesia is similar to that for cardiac operations involving CPB.
3. The left radial artery should be used to monitor arterial blood pressure, because clamping of the innominate artery maybe necessary during the procedure.

B. Aortic arch

1. Usually performed through a median sternotomy with deep hypothermic circulatory arrest. See section on DHCA.

C. Descending thoracic aorta

1. Generally performed through a left thoracotomy without cardiopulmonary bypass.
2. Monitoring
   A. Arterial blood pressure should be monitored from the right radial artery, since clamping of the left subclavian may be necessary.
   B. Pulmonary artery catheter is helpful for following cardiac function and intraoperative fluid management.
3. Cross clamping of the aorta results in a sudden increase in left ventricular afterload which may precipitate acute left ventricular failure or myocardial ischemia in patients with underlying ventricular dysfunction or coronary disease. A nitroprusside infusion is usually required to prevent excessive increases in blood pressure.
4. Release hypotension: following the release of the aortic cross clamp, the abrupt decrease in afterload combined with bleeding and the release of vasodilating acid metabolites from the ischemic lower body can precipitate severe systemic hypotension. Decreasing anesthetic depth, volume loading, and partial or slow release of the cross-clamp may help decrease the severity of hypotension.
5. Complications
   A. Paraplegia: the incidence of transient postoperative deficits (11%) and postoperative paraplegia (6%).
   B. The classic deficit is that of an anterior spinal artery syndrome with loss of motor function and pinprick sensation but preservation of vibration and proprioception.
   C. Artery of Adamkiewicz: this artery has a variable origin from the aorta, arising between T5 and T8 in 15% , between T9 and T12 in 60%, and between L1 and L2 in 25% of patients.
   D. Measures used to help protect the spinal cord include: use of a temporary heparin coated shunt or partial

cardiopulmonary bypass; mild hypothermia; mannitol (related to its ability to lower cerebrospinal pressure by decreasing its production); and drainage of cerebrospinal fluid.

E. Renal failure: infusion of mannitol (0.5 g/kg) prior to cross-clamping may decrease the incidence of renal failure. Low dose dopamine has not been shown to be as effective but may be used as an adjunct for persistently low urine output.

D. Surgery on the abdominal aorta

1. Either an anterior transperitoneal or an anterolateral retroperitoneal approach is commonly used.
2. Monitoring is similar to other aorta surgery (see above).
3. The aorta cross-clamp is usually applied to the supraceliac, suprarenal, or infrarenal aorta. In general, the farther distally the clamp is applied, the less the effect on left ventricular afterload. Heparinization is necessary prior to cross-clamp.
4. Release of the aortic clamp frequently produces hypotension. The same techniques to prevent release hypotension as discussed above should be used. Cross-clamp placed at the level of the infrarenal aorta in patients with good ventricular function frequently have minimal hemodynamic changes when the clamp is removed.
5. Fluid requirements are typically increased (up to 10-12 ml/kg/hr) because of the large incision and extensive retroperitoneal surgical dissection. Fluid requirements should be guided by central venous or pulmonary artery pressure monitoring.
6. Renal prophylaxis with mannitol should be considered, especially in patients with preexisting renal disease. Clamping of the infrarenal aorta has been shown to significantly decrease renal blood flow, which may contribute to postoperative renal failure.
7. Epidurals are commonly placed both for intraoperative and postoperative use. The combined technique of epidural and general anesthesia decreases the general anesthetic requirement and appears to suppress the release of stress hormones. Systemic heparinization increases the risk of paraplegia secondary to an epidural hematoma, however, careful placement of the epidural catheter prior to heparinization lowers the risk of an epidural hematoma.
8. Postoperative considerations are similar to other aorta surgery.

Anesthesia for Thoracic Surgery

Preoperative Laboratory Criteria for Pneumonectomy

Test	High Risk Patient
Arterial Blood Gas	$PaCO_2$ > 45 mmHg (on RA)
FEV_1	< 2 liters
FEV_1/FVC	< 50% of predicated
Maximum Breathing Capacity	< 50% of predicated
RV/TLC	< 50%

Calculation of predicted pulmonary function after pneumonectomy: Predicted postoperative FEV_1 = FEV_1 x perfusion (%) to remaining lung.

Calculation of predicted pulmonary function after lobectomy: Loss of function = preoperative FEV_1 x number of functional segments in lobe to be resected divided by the total number of segments in both lungs

1. Indications for one-lung anesthesia

A. Absolute

1. Confine pulmonary infection to one side.
2. Confine pulmonary bleeding to one side.
3. Separate ventilation to each lung.
   A. Bronchopulmonary fistula.
   B. Tracheobronchial disruption.
   C. Large lung cyst.
4. Bronchopleural lavage.

B. Relative

1. High priority

A. Thoracic aortic aneurysm.
B. Pneumonectomy.
C. Upper lobectomy.
D. Thoracoscopy.

2. Low priority

A. Middle and lower lobectomies.
B. Sub-segmental resections.
C. Esophageal surgery.

2. One-lung anesthesia

A. Physiology of one-lung anesthesia

1. One-lung anesthesia results in a large ventilation-perfusion mismatch, secondary to a large intrapulmonary shunt.
2. Factors known to inhibit hypoxic pulmonary vasoconstriction include: (1) very high or very low pulmonary artery pressures; (2) hypocapnia; (3) vasodilators; (4) high or low mixed venous oxygen; (5) pulmonary infection; (6) volatile anesthetics.
3. Factors that decrease blood flow to the ventilated lung: (1) high mean airway pressure; (2) vasoconstrictors; (3) low FIO_2.

4. Carbon dioxide elimination is usually not affected by one-lung anesthesia provide minute ventilation is unchanged.

B. Management of hypoxia during one-lung anesthesia

1. Confirm tube placement. Increase oxygen to 100%.
2. Change tidal volume (8-15 cc/kg) and ventilatory rate.
3. Periodic inflation of the collapsed lung with 100% oxygen.
4. Continuous insufflation of oxygen into the collapsed lung.
5. Adding 5 cm H_2O of continuous positive airway pressure (CPAP) to the collapsed lung.
6. Adding 5 cm H_2O of positive end expiratory pressure (PEEP) to the ventilated lung.
7. Adding additional CPAP, followed by additional PEEP.
8. Early ligation of the ipsilateral pulmonary artery (in a pneumonectomy).

3. Evaluation of lung resectability

A. Initial evaluation includes PFTs and arterial blood gas (ABG). If the $PaCO_2$ is above 40 mmHg, the maximum breathing capacity or FEV_1 is below 50%, or the residual volume/total lung capacity is greater than 50%, then split lung function tests should be performed.

B. Regional pulmonary function test

1. Regional lung function can be determined by radiospirometry.
   A. Regional perfusion (^{133}Xe, ^{131}I-MAA).
      1. Regional fractional perfusion is determined by dividing the regional perfusion washin peak by the total perfusion wash-in count.
   B. Regional ventilation ^{133}Xe.
      1. The calculation for regional fractional ventilation is similar to that for regional fractional perfusion, that is, regional counts are divided by total counts.
2. The predicted postoperative FEV1 can be calculated by multiplying the preoperative FEV1 by the percent of pulmonary function contributed by the univolved lung. the lowest predicted postoperative FEV1 that allows adequated elimination of carbon dioxide is reported to be 800 cc. Alternatively, some recommend that the predicted postoperative FEV1 be at least 40% of the predicted postoperative FEV1 based on age, sex, and size.
3. If the split lung function criteria are not satisfied balloon occlusion of the pulmonary artery ipsilateral to the diseased lung can be performed
   A. With the blood flow to the diseased lung occluded, a mean pulmonary artery pressure greater than 30-40 mmHg or a PaO2 less than 45 mmHg indicats that the patient is unable to tolerate pneumonectomy.

C. Exercising testing

1. Exercising testing has been investiged as a method of preoperative evaluation. Exercise testing not only provides an overall assessment of cardiopulmonary function but may also allow separate evaluations of cardiac and pulmonary function, however, the usefulness of preoperative exercise testing remains controversial.

Lung Transplantation

1. General inforation

A. Indications include end-stage pulmonary parenchymal disease or pulmonary hypertension.

B. Patients typically have dyspnea at rest or with minimal activity, and resting hypoxemia (PaO2 <50 mmHg) with increasing oxygen requirements.

C. Organ selection is based on size and ABO compatibility.

2. Anesthetic considerations

A. Preoperative consideration

1. These procedures are performed on an emergency basis so patients may have little time to fast for surgery.
2. Oral cyclosporine is usually given preoperatively (resulting in a full stomach).
3. Most patients are very sensitive to sedatives.

B. Intraoperative management

1. Monitoring
   A. In addtion to standard ASA monitors, arterial line, central venous pressure monitoring, and pulmonary artery catheter are commonly used.
2. Induction/Maintenance
   A. A modified rapid sequence induction with moderate head-up position is commonly utilized. Hypoxemia and hypercarbia must be avoided to prevent further increases in pulmonary artery pressure.
   B. Methylprednisolone is usually given prior to release of vascular clamps.
3. Post transplantation management
   A. Peak inspiratory pressures should be kept to the minimum compatible with good lung expansion and the inspired oxygen concentration is kept below 60%.
   B. Transplantation disrupts the neural innervation, lymphatic drainage, and bronchial circulation of the transplanted lung.
   C. Respiratory pattern is unaffected but the cough reflex is abolished below the carina.
   D. Hypoxic pulmonary vasoconstriction remains normal.
   E. Loss of lymphatic drainage increases extravascular lung water and predisposes the transplanted lung to pulmonary edema.

C. Postoperative management

1. Patients are left intubated after surgery for 24-72 hours.
2. A thoracic or lumbar epidural may be employed for postooperative analgesia when coagulation studies are normal.

Obstetrical Anesthesia

1. **Epidural/spinal analgesia for labor**
   - **A.** Epidurals generally placed after the patient is in active labor (and 4 cm dilated).
   - **B.** Patient should be well hydrated first (except PIH patients) with 500-1000 cc of crystalloid solution.
   - **C. Standard labor epidurals**
     1. Load with 8-12 cc 0.25% bupivacaine (in incremental doses) followed by a continuous infusion (several options for continuous infusion are available).
        - A. 0.125% bupivacaine at 10-14 cc/hr.
        - B. 0.0625% bupivacaine plus fentanyl 2 mcg/cc at 12-16 cc/hr.
        - C. 0.125% bupivacaine plus sufentanil 0.2 mcg/cc at 10-14 cc/hr.
        - D. 0.0625% bupivacaine plus sufentanil 0.2 mcg/cc at 12-16 cc/hr.
     2. Other options include: lidocaine (load with 10 cc of 1% lidocaine followed by continuous infusion of lidocaine 0.33% at 15 cc/hr or lidocaine 0.5% at 10 cc/hr); chloroprocaine (load with 10 cc of chloroprocaine 2.0% followed by continuous infusion of chloroprocaine 0.5% at 30 cc/hr).
     3. If additional analgesia required may add fentanyl 2 mcg/cc to infusion (usually give fentanyl bolus 50-100 mcg).
   - **D. Epidural opioids for labor**
     1. Meperidine: 100 mg provides good relief for about 2-3 hours.
     2. Fentanyl: 100-200 mcg provides quick onset (5-10 min) but brief duration (1-2 hours).
     3. Sufentanil: 5-15 mcg (mixed in 10 cc preservative free saline) provides good relief for about 1-2 hours.
   - **E. Intrathecal opioids for labor**
     1. Possible role of intrathecal opioids include multips in very active labor (6-9 cm) or primips that are fully dilated with significant pain. Can also be used for patients in early labor (2-4 cm dilated, prior to active phase).
     2. Fentanyl: 25 mcg fentanyl in 1 cc preservative free saline (provides about 1 hour of analgesia).
     3. Meperidine: 10-20 mg (provides about 2 hours of analgesia).
     4. Sufentanil: 10 mcg in 1-2 cc preservative free saline (provides about 3 hours of analgesia).
   - **F. Combined spinal-epidural for labor**
     1. Possible role of combined spinal/epidural technique include patients presenting in early labor (ie, the spinal can be given to help with early labor pain, while the epidural can be activated after the patient is in active labor and her pain returns).
     2. Spinal: 25 mcg fentanyl in 1 cc saline or 10 mcg sufenta in 1 cc saline.
     3. Epidural: as noted above (started after pain returns).

2. Analgesia for assisted vaginal delivery (sitting dose)

A. Epidural

1. Prehydrate, then use 2% or 3% chloroprocaine 10-15 cc with patient in at least head up 45 degrees, expect level to rise to about T8-T10.

B. Spinal

1. Prehydrate, use 40-50 mg of hyperbaric 5% lido in sitting position.

3. Cesarean sections

A. General notes

1. All patients should receive bicitra 30 cc (metoclopramide 10 mg IV optional).
2. All patients should have a wedge under the right hip for left uterine displacement (15 degrees).
3. Pre-op labs: hct, hb, clot to blood bank (for crash cesarean section hct).
4. Patients with PIH should have PT/PTT, platelet, and bleeding time done prior to epidural/spinal.
5. Treat hypotension with fluids and ephedrine to maintain systolic blood pressure >100.
6. After placenta delivered: oxytocin 20-40 units added to IV fluids (if the uterus does not contract readily, methergine, 0.2 mg IM, may be given).

B. Epidural anesthesia

1. Initial dose: 1.5-2% lidocaine with/without epi, 0.5% bupivacaine or 3% chloroprocaine may be used (most commonly use 20-25 cc 2% lidocaine with epi). Administer approximately 20 cc (in small increments) to obtain T2-T4 level. Rebolus as necessary.
2. Fentanyl: 50-100 mcg has been found to speed the onset, potentiate intraoperative analgesia, decrease nausea and vomiting during uterine manipulation, decrease requirements for supplemental opioid medication, reduce shivering, with no adverse maternal or neonatal effects.
3. Post-op pain control: duramorph 3-5 mg epidurally, given after the baby is delivered provides 18-24 hours of postoperative pain relief.

C. Spinal anesthesia

1. Anesthetic Agent	**Dose**	**Duration**	**Onset**
lidocaine	50-75 mg	30-45 (min)	2-3 (min)
tetracaine	8-10 mg	90-180	4-6
bupivacaine	8-15 mg	90-120	2-4

2. Fentanyl: 10-25 mcg has been found to speed the onset, potentiate intraoperative analgesia, decrease nausea and vomiting during uterine manipulation, decrease requirements for supplemental opioid medication, reduce shivering, with no adverse maternal or neonatal effects**.**

3. Duramorph: 0.10-0.25 mg provides 18-24 hours of postoperative pain relief (onset in 60 minutes).

D. Patchy blocks: prior to delivery of the baby can be treated with ketamine, 10-20 mg IV, or 30% nitrous; after delivery, IV narcotic may be used.

E. Crash cesarean sections

1. Induction: preoxygenate, cricoid pressure, thiopental 4 mg/kg (ketamine 1 mg/kg for asthmatics and hemodynamically unstable patients), succinylcholine 1.5 mg/kg, nitrous 50%/oxygen 50%, volatile agent (enflurane 0.5-0.75% or isoflurane 0.75%).
2. After baby delivered: may add muscle relaxant (usually one dose of atracurium, 0.5 mg/kg, or vecuronium, 0.05 mg/kg), fentanyl 100-150 mcg, versed 1-2 mg, nitrous 70%/oxygen 30%, and discontinue inhalation agent.

4. Pain pathways

A. Pain during the first stage of labor is due to uterine contractions and cervical dilatation and is carried by the visceral afferent fibers (T10 to L1).

B. Pain at the end of the first stage signals the beginning of fetal descent. Second stage of labor is due to stretching of the birth canal, vulva, and perineum and is conveyed by the afferent fibers of the posterior roots of the S2 to S4 nerves.

5. Pregnancy induced hypertension (PIH)

A. 5-7% incidence in pregnancy occurring mostly in young primigravidas attributed to lack of prenatal care.

B. Predisposing factors: multiple gestation, major uterine anomalies, chronic hypertension, chronic renal disease, diabetes, polyhydramnios, molar pregnancy, fetal hydrops.

C. Consists of hypertension (SBP >140 or DBP >90 or a consistent increase in SBP by 30 or DBP by 15, proteinuria (> 500 mg/day), and generalized edema occurring after the 20th week of gestation and resolving within 48 hours after delivery.

D. Severe PIH is defined as BP > 160/110, pulmonary edema, proteinuria >5 gm/day, oliguria, central nervous system manifestations, hepatic tenderness, or HEELP syndrome; causes of death include pulmonary edema and cerebral hemorrhage.

E. Pathophysiologic alterations

1. **Hematologic:** decrease in intravascular volume (primarily plasma), disseminated intravascular coagulation characterized initially by reduction in platelets; later by rise in fibrin degradation products, fall in fibrinogen level, increased PT/PTT.
2. **Cerebral:** hyperreflexia, CNS irritability increase, coma, increased intracranial pressure, altered consciousness.
3. **Respiratory:** upper airway and laryngeal edema.
4. **Cardiac:** arteriolar constriction and increase of peripheral resistance leading to increased BP.
5. **Ophthalmic:** retinal arteriolar spasm, possible bilateral retinal detachment owing to massive retinal edema, and blurred vision.
6. **Renal:** reduction in renal blood flow and GFR, elevated plasma uric acid (increased levels correlate with severity of disease), deposition of fibrin in glomeruli.
7. **Uterine:** hyperactive uterus, markedly sensitive to oxytocin; placenta may show signs of premature aging, infarcts, fibrin deposits, calcifications and/or abruption.

8. **Hepatic:** elevated LFT's, hepatocellular damage or edema secondary to vasospasm, epigastric or right upper quadrant abdominal pain.

F. Treatment

1. Definitive therapy includes delivery of fetus and the placenta.
2. Hospitalization, bed rest in lateral decubitus position, fluids should not be restricted.
3. Antihypertensive drugs (hydralazine). In a crisis continuous infusions of trimethaphan, sodium nitroprusside, or nitroglycerin can be used.
4. Magnesium therapy: therapeutic maternal blood levels of 4-8 meq/liter. Give 2-4 gram loading dose (slow IV over 5-15 minutes) followed by continuous infusion of 1-3 grams/hour.
5. Fluid replacement colloid more effective than crystalloid in correcting hypovolemia (isotonic crystalloid or LR solution at a rate of 75-150 cc/hr until urine output of 20-30 cc/hr).

G. Anesthetic management

1. All patients: bleeding time, platelet count, coagulation profile, CBC, Mg level, fibrinogen, fibrin split products, lytes, uric acid level, LFT's.
2. Indications for invasive monitoring
   A. Unresponsive or refractory hypertension: increased systemic vascular resistance or increased cardiac output.
   B. Pulmonary edema: Cardiogenic or left ventricular failure, increased systemic vascular resistance, or noncardiogenic volume overload.
   C. Persistent arterial desaturation.
   D. Oliguria unresponsive to modest fluid loading: low preload, severe increased systemic vascular resistance with low cardiac output, selective renal artery vasoconstriction.
3. Other orders and medications: patients should have blood pressure under control before (DBP<110) starting epidural. Epidural anesthesia is the preferred method of analgesia for vaginal delivery and cesarean section in most patients including those with eclampsia. Spinal can be used. General anesthetics are reserved for fetal distress, coagulopathies or hypovolemia.

H. HELLP Syndrome

1. HELLP syndrome: hemolysis, elevated liver enzymes, low platelet counts.
2. Occurs in 4-12% of severe pregnancy induced hypertensive patients.
3. Reported perinatal mortality: 7.7-60%. Maternal mortality 3.5-24.2%.
4. Diagnostic criteria: platelet count less than 100,000/mm^3, hemolysis by peripheral smear and increased bilirubin (greater than 1.2 mg/dl, elevated SGOT (greater than 70 U/L) and LDH (greater than 600 U/L).
5. High incidence of maternal complications including abruptio placenta, coagulopathy (DIC, prolonged PT and PTT), acute renal failure, ruptured hepatic hematoma.

6. **Antepartum hemorrhage**
   - **A. Placenta previa:** abnormal implantation of the placenta in the lower uterine segment; incidence: 0.1-1.0% (higher in subsequent pregnancies); presents with painless vaginal bleeding; potential for massive blood loss; risk factors include prior uterine scar, prior placenta previa, advanced maternal age, and multiparity.
   - **B. Abruptio placentae:** premature separation of a normally implanted placenta; incidence: 0.2-2.4% (predisposing conditions: hypertension, uterine abnormalities, history of cocaine abuse); presents with painful vaginal bleeding, abnormalities in fetal heart rate, irritable uterus; potential for massive blood loss (blood loss may be concealed), disseminated intravascular coagulation (DIC), renal failure.
   - **C. Uterine rupture:** incidence: 0.008-0.1% (predisposing conditions: previous uterine surgery, prolonged intrauterine manipulation); presents with pain with or without vaginal bleeding, abnormalities in fetal heart rate, irritable uterus; potential for massive blood loss.
7. **Physiological changes in pregnancy**
   - **A. Hematological alterations**
     1. Increased plasma volume (40-50%).
     2. Increased total blood volume (25-40%).
     3. Dilutional anemia (hematocrit 35%).
   - **B. Cardiovascular changes**
     1. Increased cardiac output (30-50%).
     2. Aortocaval compression (supine hypotension syndrome occurs in 10%).
   - **C. Ventilatory changes**
     1. Increased alveolar ventilation (70%).
     2. Decreased functional residual capacity (20%).
     3. Airway edema.
     4. Decreased $PaCO_2$ (30%).
   - **D. Gastrointestinal changes**
     1. Prolonged gastric emptying.
     2. Decreased lower esophageal sphincter tone.
   - **E. Altered drug responses**
     1. Decreased requirements for inhaled anesthetics (MAC).
     2. Decreased local anesthetic requirements.
8. **Biophysical monitoring**
   - **A. Early deceleration:** related to head compression.
   - **B. Late deceleration:** related to uteroplacental insufficiency.
   - **C. Variable deceleration:** related to umbilical cord compression.

Post-cesarean Section Epidural Opioid Analgesia

Drug	Recommended Dose	Onset Time (min)	Duration (hr)
Morphine	3-5 mg	30-90	12-18
Fentanyl	50-100 mcg/10 cc NS	5-10	2-3
Sufentanil	25-30 mcg	3-5	2-4
Butorphanol	1-2 mg/10 cc NS	10-15	3-5

Post-cesarean Section Subarachnoid Opioid Analgesia

Drug	Recommended Dose	Onset Time (min)	Duration (hr)
Morphine	0.1-0.3 mg	30-45	12-15
Fentanyl	10-20 mcg	5-10	2-4

IV Morphine PCA Protocol for Post-cesarean Section Analgesia

Drug	Recovery Room	Ward	Complaint of Pain
Bolus	5 mg		0.1 mg/kg
Dose	5 mg	1 mg	2 mg
Lockout	5 min	8-10 min	8-10 min
Basal Rate	1 mg/hr	1 mg/hr	1 mg/hr
Limit	30 mg/hr	7 mg/hr	13 mg/hr

Fentanyl PCEA Protocol for Post-cesarean Section Analgesia

Drug	Recovery Room	Ward	Complaint of Pain
Bolus	100 mcg		50-100 mcg
Dose	40 mcg	40 mcg	50-60 mcg
Lockout	10 min	10 min	10 min
Basal Rate	60 mcg/hr	60 mcg/hr	60-80 mcg/hr
Limit	260 mcg/hr	260 mcg/hr	310-380 mcg/hr

Sufentanil PCEA Protocol for Post-Cesarean Section Analgesia

Drug	Recovery Room	Ward	Complaint of Pain
Bolus	30 mcg		20 mcg
Dose	8 mcg	4 mcg	8 mcg
Lockout	10 min	10 min	10 min
Basal Rate	6 mcg/hr	6 mcg/hr	6 mcg/hr
Limit	46 mcg/hr	26 mcg/hr	46 mcg/hr

Neuroanesthesia

Comparative Effects of Anesthetic Agents

Agent	CMR	CBF	DVD	CSF Pro	CSF Abs	CBV	ICP
Halothane	--	+++	Yes	0/-	-	++	++
Enflurane	--	++	Yes	+	-	++	++
Isoflurane	---	+	Yes	0	+	++	+
Desflurane	----	+		+	-	?	++
Sevoflurane	---	+		?	?	?	++
Nitrous Oxide	+	+	?	0	0	0	+
Ketamine	++	++	Yes	0	-	++	++
Barbiturates	---	---	No	0	+	--	---
Etomidate	---	--	No	0	+	--	--
Propofol	--	--	No	?	?	--	--
Benzo-diazepines	-	-	No	0	+	-	-
Narcotics	0	0		0	+	0	0
Droperidol	0	0		?	?	0	0
Lidocaine	--	--		?	?	--	--
Succinyl-choline	+						+

Key: + = increase; - = decrease; 0 = no change; ? = Unknown; CMR = cerebral metabolic rate; CBF = cerebral blood flow; DVD = direct vasodilation; CSF Pro = CSF production; CSF Abs = CSF absorption; CBV = cerebral blood volume; ICP = intracranial pressure.

Basic Neurophysiology

1. Cerebrospinal fluid (CSF)

A. Produced at a rate of 0.3 cc/min (about 21 cc/hr or 500 cc/day). CSF is produced primarily be the choroid plexuses of the cerebral (mainly lateral) ventricles. Smaller amounts are formed directly by ependymal cell linings and yet smaller amounts from fluid leaking into the perivascular spaces surrounding cerebral vessels (blood-brain barrier leakage). CSF is reabsorbed at a rate of 0.3-0.4

cc/min into the venous system by the villi in the arachnoid membrane.

B. CSF production is decreased by carbonic anhydrase inhibitors (acetazolamide), corticosteroids, spironolactone, loop diuretics (furosemide), isoflurane, and vasoconstrictors.

C. Cerebral spinal fluid volume: 100-150 ml normal.

2. Cerebral blood flow

A. Cerebral blood flow averages 50 ml/100 gm/min (gray matter is about 80 ml/100 gm/min and white matter is about 20 ml/100 gm/min).

B. Cerebral blood flow rates

1. Total cerebral blood flow in adults averages 750 cc/min (15-20% of cardiac output).
2. Flow rates below 20-25 cc/100 g/min are usually associated with cerebral impairment (slowing of EEG).
3. Cerebral blood flow rates between 15 and 20 cc/100 g/min produce a flat (isoelectric) EEG.
4. Cerebral blood flow rates below 10 cc/100 g/min are usually associated with irreversible brain damage.

C. Cerebral blood flow determinants

1 $PaCO_2$ for every 1 mmHg change in $PaCO_2$ from there is a corresponding change in CBF by 1 ml/100 g/min.
2. Temperature: CBF is reduced 7% for every degree Celsius below 37°.
3. PaO_2: no significant increase in CBF until below 50 mmHg.
4. Cerebral perfusion pressure autoregulation: CPP = MAP - CVP (or ICP if greater), chronic hypertension shifts the autoregulation curve to the right; autoregulation is impaired in presence of intracranial tumors or volatile anesthetics.
5. Anesthetic drugs: volatile agents are potent cerebral vasodilators (halothane > enflurane > isoflurane), ketamine and nitrous oxide are cerebral vasodilators (opioids, benzodiazepines, and barbiturates are cerebral vasoconstrictors).
6. Hematocrit: CBF increases with decreasing viscosity (hematocrit). Optimal cerebral oxygen delivery occurs at hematocrits between 30-34%.
7. Regionally, CBF and metabolism are tightly coupled. An increase in cortical activity will lead to a corresponding increase in CBF.
8. Sympathetic tone does not appreciably affect CBF.
9. Intracranial components are brain bulk (80%), blood volume (5%), and CSF (15%). A small increase in intracranial volume can be partially compensated by translocation of CSF into the spinal subarachnoid space and compression on the venous blood volume. This compensation mechanism is limited, and once exhausted, any further increase in volume will lead to a rise in ICP.

3. Cerebral metabolism

A. The brain receives 15% of the cardiac output and consumes 20% of the oxygen.

B. Cerebral metabolic rate for oxygen averages 3.0-3.5 ml/100 gm/min. Cerebral metabolic rate ($CMRO_2$) is greatest in the gray matter of

the cerebral cortex and generally parallels cortical electrical activity.

C. Luxury perfusion: the term describing the combination of a decrease in neuronal metabolic demand with an increase in cerebral blood flow (metabolic supply). This altered coupling of cerebral blood flow and cerebral metabolic rate occurs with volatile agents.

4. Intracranial pressure (ICP)

A. Normal ICP is 5-10 mmHg.

1. Intracranial hypertension is defined as a sustained increase in ICP above 15 mmHg.
2. When intracranial pressure exceeds 30 mmHg, cerebral blood flow progressively decreases and a vicious cycle is established: ischemia causes brain edema, which in turn increases intracranial pressure, resulting in more ischemia.
3. Periodic increases in arterial blood pressure with reflex slowing of the heart rate (Cushing response) are often observed and can be correlated with abrupt increases in intracranial pressure lasting 1-15 minutes.

B. Cerebral perfusion pressure (CPP) = MAP - ICP (or central venous pressure (CVP), whichever is greater).

C. Compensatory mechanisms for increased ICP

1. Displacement of CSF from the cranial to the spinal compartment.
2. Increase in CSF absorption.
3. Decrease in CSF production.
4. Decrease in total cerebral blood volume (primarily venous).

D. Methods of decreasing ICP

1. Reduce cerebral blood volume

A. Hyperventilation ($PaCO_2$ 20-25 mmHg). Excessive hypertventilation ($PaCO_2$ <20) may cause cerebral ischemia.

B. Prevent straining or coughing on the endotracheal tube.

C. Elevation of the head to encourage venous drainage.

2. Reduce cerebrospinal fluid volume

A. Drain through ventriculostomy or lumbar subarachnoid catheter.

B. Decrease CSF production with acetazolamide.

C. Recent studies suggest that administration of hypertonic saline and mannitol reduce the production of CSF and may contribute to the immediate effect of ICP reduction.

3. Reduce brain volume (brain bulk)

A. Decrease brain water with osmotic diuretics (20% mannitol 0.25 - 1.0 g/kg); mannitol is thought to reduce cerebral swelling by osmotic dehydration, loop diuretics (furosemide 0.5 mg/kg), steroids (decadron), and other drugs (barbiturates, etomidate, propofol).

5. Evidence of increased intracranial pressure

A. Symptoms: nausea or vomiting, mental status changes (drowsiness progressing to coma), personality changes, visual changes, neck stiffness, focal deficits, hypertension, bradycardia, absent brain stem reflexes, decerebrate posturing, fixed and dilated pupils,

respiratory rhythm changes (irregular rhythm or apnea).

B. Signs: headache, papilledema, posturing, bulging fontanelles in infants, seizures, altered patterns of breathing, cushing's reflex (hypertension and bradycardia).

C. Radiologic Signs

1. X-ray: suture separation, erosion of clinoid process, copper-beaten skull.
2. CT/MRI Scans: midline shift, cerebral edema, mass lesions, abnormal ventricular size, obliteration of basal cistern.

D. Cushing reflex

1. Cushing reflex: periodic increases in arterial blood pressure with reflex slowing of the heart is the Cushing response. The Cushing response is often observed and can be correlated with abrupt increases in intracranial pressure (plateau or A waves) lasting 1-15 minutes.
2. Cushing triad: hypertension, bradycardia, respiratory disturbances (late and unreliable sign that usually just proceeds brain herniation).
3. Continued profound sympathetic nervous system (SNS) discharge during Cushing's reflex may hide a state of hypovolemia. If the Cushing source is taken away by surgical intervention and/or the SNS response is ablated by anesthesia, one may encounter profound and resistant hypotension.

6. Cerebral blood flow determinants (normal CBF 50 ml/100 g/min)

A. $PaCO_2$: for every 1 mmHg change in $PaCO_2$ from there is a corresponding change in CBF by 1 ml/100 g/min.

B. Temperature: CBF is reduced 7% for every degree Celsius below 37°.

C. PaO_2: no significant increase in CBF until below 50 mmHg.

D. Cerebral perfusion pressure autoregulation: CPP = MAP - CVP (or ICP if greater), chronic hypertension shifts the autoregulation curve to the right; autoregulation is impaired in presence of intracranial tumors or volatile anesthetics.

E. Anesthetic drugs: volatile agents are potent cerebral vasodilators (halothane > enflurane > isoflurane), ketamine and nitrous oxide are cerebral vasodilators (opioids, benzodiazepines, and barbiturates are cerebral vasoconstrictors).

F. Hematocrit: CBF increases with decreasing viscosity (hematocrit).

7. Potential methods of CNS protection

A. Decrease metabolism: etomidate, isoflurane, barbiturates.

B. Hypothermia

1. Hypothermia is the most effective method for protecting the brain during focal and global ischemia.
2. Hypothermia decreases both basal and electrical metabolic requirements throughout the brain.

C. Decrease secondary injury: glucose control, anticonvulsants, steroids, calcium entry blockers.

D. Increase CBF: hypertension, hemodilution, anticoagulation, rheologic agents.

Practical Clinical Points

1. General issues for patients scheduled for craniotomy for mass lesions

A. Preoperative

1. Preanesthetic evaluation should attempt to establish the presence or absence of intracranial hypertension. Laboratory evaluation should rule out corticosteroid-induced hyperglycemia and electrolyte disturbances due to diurectics or abnormalities in antidiurectic hormone secretion.
2. Examination should include a neurologic assessment documenting mental staus and any existing existing sensory or motor deficits.
3. Computerized tomography (CT) and MRI scans should be reviewed for evidence of brain edema, a midline shift greater than 0.5 cm, and ventricular size.

B. Premedication

1. Premedication is best avoided when intracranial hypertension is suspected.
2. Corticosteroids and anticonvulsant therapy should be continued up until the time of surgery.

C. Monitoring

1. In addition to standard ASA monitors, direct intra-arterial pressure monitoring and bladder catheterization are mandatory for most patients undergoing craniotomy.
2. Central venous access and pressure monitoring should be considered for patients requiring vasoactive drugs. Use of the internal jugular vein for access is somewhat controversial because of the risk of carotid puncture and concern that the catheter might interfere with venous drainage from the brain.

Anesthetic Management in Traumatic Brain Injury

1. Preoperative assessment

A. The initial examination must include an expeditious search for other injuries. The Glasgow Coma Scale (GCS) is accepted for the evaluation of level of consciousness in patients with head injury.

B. As with other trauma patients, all head injury patients should be considered to have a full stomach.

C. Patients with obvious hypoventilation, an absent gag reflex, or GCS score below 8 require tracheal intubation and hyperventilation.

D. Dysrhythmias and electrocardiographic abnormalities in the T wave, U wave, ST segment, and QT interval are common following head injuries but are not necessarily associated with carediac injury; they likely represent altered autonomic function.

E. See section on trauma anesthesia for additional information.

2. Induction and maintenance of anesthesia

A. In patients who have a normal airway and are hemodynamically stable, a rapid sequence induction of anestheisa with thiopental 3-4 mg/kg, lidocaine 1-1.5 mg/kg, and succinylcholine or other muscle relaxant is indicated. The use of succinylcholine is controversial, however, the minimal risks associated with succinylcholine must be balanced against the damaging effects of hypoxemia and hypercapnia as well as straining and coughing on the brain.

B. In hemodynamically unstable patients, etomidate 0.2 -0.3 mg/kg should be used. Thiopental and propofol are relatively contraindicated in hypovolemic patients.

C. There is no ideal method of treating a head-injured patient with a difficult airway. In a stable cooperative patient the airway can be anesthetized topically and direct or fiberoptic laryngoscopy undertaken. In an unstable patient, retrograde intubation is an option, but tracheotomy may be required. Nasal intubations are contraindicated in patients with basilar skull fracture.

D. Anesthesia is usually maintained with a combination of a barbiturate, opioid volatile agent and muscle relaxant.

3. Other information

A. Disseminated intravascular coagulation may be seen with severe head injuries secondary to release of large amounts of brain thromboplastin.

Pain Management

Pain Management: Patient Controlled Analgesia (PCA)

Drug	Bolus Dose (mg)	Lockout Interval (min)	Continuous Infusion (mg/hr)
Agonists			
Fentanyl	0.015-0.05	3-10	0.02-0.1
Hydromorphone	0.1-0.5	5-15	0.2-0.5
Meperidine	5-15	5-15	5-40
Methadone	0.5-3.0	10-20	
Morphine	0.5-3.0	5-20	0.5-10
Oxymorphone	0.2-0.8	5-15	0.1-1.0
Sufentanil	0.003-0.015	3-10	0.004-0.03
Agonist-Antagonists			
Buprenorphine	0.03-0.2	10-20	
Nalbuphine	1-5	5-15	1-8
Pentazocine	5-30	5-15	6-40

Patient Controlled Epidural Analgesia (PCEA)

Drug Mixture	Loading Bolus (ml)	Dose (ml)	Lockout (min)	Continuous Infusion (ml/hr)
Fentanyl 2 mcg/cc + Bupivacaine 0.125%	4-12	2-4	10	2-8
Fentanyl 5 mcg/cc + Bupivacaine 0.0625%	6-12	2-6	10	2-8
Sufentanil 2 mcg/cc + Bupivacaine 0.0625%	4-10	2-4	10	2-4
Fentanyl 10 mcg/cc + Bupivacaine 0.05%	5-10	2-5	10	2-5
Fentanyl 10 mcg/cc + Bupivacaine 0.025%	5-10	2-5	10	2-5
Hydromorphone 0.04 mg/cc	5-15	0.5-4.0	20	0-2
Morphine 0.1 mg/cc	10-20	0.5-2.0	20	0-4

Intrathecal (Spinal) Opioids

Opioid	Dose	Onset (min)	Duration (hr)
Morphine	0.15-0.6 mg	15-45	8-24
Fentanyl	10-25 mcg	2-5	1-3
Sufentanil	5-15 mcg	2-5	2-4

Epidural Opioids for Postoperative Analgesia

Drug	Bolus Dose	Onset (min)	Peak (min)	Duration (hr)	Concen-tration	Rate (ml/hr)
Meperidine	30-100 mg	5-10	12-30	4-6	1 mg/cc	10-20
Morphine	5 mg	23.5	30-60	12-24	0.1 mg/cc	1-6
Methadone	5 mg	12.5	17	7.2		
Hydro-morphone	1 mg	13	23	11.4	0.05 mg/cc	6-8
Fentanyl	100 mcg	4-10	20	2.6	4 mcg/cc	4-12
Dia-morphine	5 mg	5	9-15	12.4		
Sufentanil	30-50 mcg	7.3	26.5	3.9		
Alfentanil	15 mcg/kg	15		1-2		

Continuous Epidural Infusion Analgesia

Bupivacaine (%)	Opioid (concentration)	Infusion Rate (ml/hr)
0.125	Fentanyl 2 mcg/cc	4-8
0.125	Sufentanil 1 mcg/cc	4-8
0.125	Morphine 0.05 mg/cc	4-8
0.0625	Fentanyl 5 mcg/cc	4-10
0.0625	Sufentanil 2 mcg/cc	4-8
0.0625	Morphine 0.1 mg/cc	2-8
	Morphine 0.1 mg/cc	2-8
	Hydromorphone 0.02 mg/cc	1-6

Initial Dose (mg) of Epidural Morphine for Treatment of Acute Pain

Patient Age (yrs)	Nonthoracic Surgery (lumbar cath)	Thoracic Surgery (thoracic cath)	Thoracic Surgery (lumbar cath)
15-44	4	4	5
45-65	3	3	4
66-75	2	2	3
76+	1	1	2

Pain Management: Selected Opioid Analgesic Preparations

Drug	Dose (Adult)
Butorphanol Tartrate (Stadol)	1-2 mg IV q3-4 hr or 2 mg IM q3-4 hr
Codeine	15-60 mg po q4-6 hr
Hydromorphone HCL (Dilaudid)	2 mg q4-6 hr po or 1-2 mg q4-6 hr SC/IM
Levorphanol Tartrate	2 mg q6-8 hr po or SC
Meperidine HCL (Demerol)	25-150 mg q3-4 hr po, SC, or IM
Morphine (MS Contin)	30-60 mg q12 hr po
Nalbuphine HCL (Nubain)	10 mg q3-4 hr SC, IM, or IV
Oxycodone HCL	5 mg q6 hr po
Oxymorphone (Numorphan)	1-1.5 mg q4-6 hr SC or IM
Pentazocine HCL (Talwin NX)	1 tab q3-4 hr po
Propoxyphene HCL (Darvon)	65 mg q4 hr po (cap)

Pain Management: Nonsteroidal Anti-Inflammatory Drugs

Generic Name	Dose	Comments
Acetaminophen	325-650 mg q4-6h p.o.	Indicated in hemostatic problems, gastric ulcers, or gouty arthritis; hepatic toxicity follows large doses
Aspirin	325-650 mg q4-6h p.o.	May cause dyspepsia and GI bleeding, decrease platelet function; less hepatic and renal toxicity
Diflunisal	200-500 mg q8-12h p.o.	Lessirritating to GI tract
Ibuprofen	200-400 mg q8-12h p.o.	Fewer GI symptoms
Indomethacin	25-50 mg q8h p.o.	Not recommended in chronic benign pain
Ketorolac	10 mg q6h p.o. (see pharm section for IV dosing	For acute exacerbations of chronic pain; controversial for longtime use
Naproxen	500 mg initially, then 250 mg q6-8h p.o.	Slightly more toxic than ibuprofen regarding GI and CNS effects
Phenylbutazone	100 mg q6h p.o.	Can cause aplastic anemia, hepatic and renal tubular necrosis; not for chronic pain
Piroxicam	200 mg o.d.	Not recommended for chronic benign pain
Sulindac	150-200 mg q12h p.o.	Less GI toxicity; not recommended in chronic pain

Postoperative Pain Management

Potential benefits of epidural analgesia

1. A. Superior pain relief, decreased incidence of pulmonary complications, decreased incidence of cardiovascular complications, attenuated neuroendocrine/metabolic response to surgical stress, lower incidence of deep-vein thrombosis and vascular graft occlusion, earlier return of bowel function, decreased time on ventilator after major surgery or chest trauma, shorter postoperative stay in ICU.

2. Clinical pharmacology of epidural opioids

A. Hydrophilic opioids (morphine, hydromorphone)

1. Properties: slow onset, long duration, high CSF solubility, extensive CSF spread.

2. Advantages: prolonged single-dose analgesia, thoracic analgesia with lumbar administration, minimal dose compared to IV administration.
3. Disadvantages: delayed onset of analgesia, unpredictable duration, higher incidence of side effects, delayed respiratory depression.

B. Lipophilic opioids (fentanyl, sufentanil)
1. Properties: rapid onset, short duration, low CSF solubility, minimal CSF spread.
2. Advantages: rapid analgesia, decreased side effects, ideal for continuous infusion or PCEA.
3. Disadvantages: systemic absorption, brief single-dose analgesia, limited thoracic analgesia with lumbar administration.

3. Optimal epidural placement for postop local anesthetic administration
A. Thoracotomy: T4-T6.
B. Upper abdominal/flank: T8.
C. Lower abdominal: T10-T12.
D. Lower extremity/pelvic: L2-L4.

4. Side effects of peridural administered opioids
A. Nausea/vomiting: opioids in the vomiting center and the chemoreceptor trigger zone in the medulla can cause nausea or vomiting.
B. Pruritus: although histamine release may play a small role, the cause of pruritus is unknown.
C. Respiratory depression
1. Patients at risk for respiratory depression are the elderly; patients who receive concomitant systemic opiates or sedatives; and patients who have received large doses of spinal opiates.
2. Early respiratory depression can occur within two hours of spinal opioid administration and is similar to that observed with parenteral administration of an opioid. With hydrophilic agents (i.e., morphine), late respiratory depression commonly peaks at 12 or 13 hours after the initial dose but can occur as late as 24 hours.
3. Epidural or spinal opiates for postoperative analgesia are contraindicated when there is lack of adequate nursing education in the care and monitoring of patients who have received epidural or spinal opiates.
D. Urinary retention.
E. Delayed gastric emptying.

5. Management of opioid related side effects
A. Nausea/vomiting
1. Metoclopramide (reglan): 10-20 mg IV q4 hrs; low incidence of side effects.
2. Droperidol (inapsine): 0.625 mg IV q4 hrs; can cause dysphoria, hypotension.
3. Scopolamine patch: 1.5 mg transdermal patch q3 hrs; effective for motion-related nausea; best used prophylactically; delayed onset (>4 hrs).
4. Ondansetron (zofran): 2-4 mg IV q6 hrs; no associated sedation.

B. Pruritus

1. Naloxone (narcan): 10-40 mcg/hr IV continuous infusion; will not significantly reverse analgesia at recommended doses.
2. Nalbuphine (nubain): 5-10 mg IV q4 hrs; may improve analgesia; may cause sedation.
3. Butorphanol (stadol): 1-2 mg IV q4 hrs; may improve analgesia; may cause sedation and dysphoria.
4. Diphenhydramine (benadryl): 25-50 mg IV q4 hrs; significant sedative effect.

C. Respiratory depression

1. Naloxone (narcan): 40-100 mcg/bolus titrated q2-3 minutes; larger than necessary dosage may result in significant reversal of analgesia, nausea, vomiting, sweating, and/or circulatory stress.

D. Urinary retention

1. Foley as needed.

Trauma Anesthesia

1. **Initial assessment: A**irway, **B**reathing, **C**irculation (fluid resuscitation).
   A. Airway: see below (trauma airway management).
2. **Secondary survey:** the patient is more extensively examined, etc.
3. **Indications for immediate endotracheal intubation:** obvious intracranial injury, GCS of less than 9, the presence of severe shock or cardiac arrest, obvious flail chest injury with paradoxical chest wall motion.
4. **Intraoperative management: general principles**
   A. Two functioning large bore IVs before induction .
   B. If possible, blood should be available (in the room) before incision is made.
   C. Induction
      1. All trauma patients are full stomachs.
      2. When general anesthesia is planned, rapid sequence induction with cricoid pressure is the method of choice.
      3. Which induction agent or whether or not drugs are used for induction depends on severity of the injury. Reduced doses of induction agent or no induction agent are at times appropriate in severely injured, obtunded patients.
   D. Maintenance
      1. Narcotic based anesthetic is the most common.
      2. For the unstable patient, scopolamine/oxygen/pancuronium can be used as a last resort (titrate a volatile anesthetic or narcotics as soon as feasible). To minimize hypotension, the depth of anesthesia may be insufficient to prevent intraoperative awareness or recall. Prophylactic use of scopolamine (0.1-0.2 mg IVP) or midazolam (1-3 mg IVP) may be considered.
      3. The key to the safe management of shock patients is to administer small incremental doses of whichever agents are selected.
   E. Avoid using nitrous oxide.
   F. Keep patient warm. All patients should have a blanket warmer, fluid warmer, and bair hugger (bair hugger should be either on the upper body or lower body). Hypothermia worsens acid-base disorders, coagulopathies (platelet sequestration and red blood cell deformities), and myocardial function.
   G. Prolonged use of anesthetics associated with sympathetic nervous system stimulation should be avoided. Continued need for vasopressors should be interpreted as hypovolemia or cardiac tamponade until proven otherwise.
   H. Recognize that blood pressure is an unreliable index of blood volume, cardiac output, and tissue perfusion.
   I. Acid-base imbalances generally respond to hydration and improved organ perfusion. Sodium bicarbonate, which dissociates into bicarbonate ion and CO_2, may temporarily worsen intracellular acidosis because cell membranes are relatively insoluble to bicarbonate compared to CO_2

5. Anesthetic considerations

A. Head trauma

1. Check Glasgow Coma Scale (see below).
2. Head injury signs: altered consciousness, restlessness, convulsions, cranial nerve dysfunction.
3. Cushing triad: hypertension, bradycardia, respiratory disturbances (late and unreliable sign that usually just proceeds brain herniation).
4. Intubation is required for airway protection and hyperventilation. Hyperventilation serves to decrease cerebral blood flow by causing cerebral vasoconstriction via an increase in intracellular pH. The reduction in cerebral blood flow results in a concomitant decrease in intracranial pressure.
5. Intubate with thiopental (or etomidate), lidocaine, succinylcholine, consider narcotics.
6. Elevated ICP: controlled by fluid restriction (except in hypovolemic shock), diuretics (mannitol), steroids, hyperventilation ($PaCO_2$ around 25 mmHg).
7. Hypotension should be avoided because of the increased risk of regional cerebral ischemia. In general, cerebral perfusion pressure (the difference between mean arterial at the level of the brain and the larger of central venous pressure or intracranial pressure) should be maintained above 60 mmHg.

B. Spinal trauma

1. Lesions involving phrenic nerve (C3-C5) usually result in apnea.
2. Loss of intercostal function: limits pulmonary reserve, ability to cough; requiring intubation; T7 or higher is the critical level for significant alveolar ventilation impairment.
3. Lesions involving high thoracic (T1-T4) may lead to bradycardia.
4. Spinal shock: seen in high spinal cord injuries lasting from a few hours to several weeks; characterized by loss of sympathetic tone in capacitance and resistance vessels below the level of the lesion; flaccid paralysis; total absence of visceral and somatic sensation below level of injury; paralytic ileus; loss of spinal cord reflexes below level of injury.
5. Autonomic hyperreflexia: associated with lesions above T5, not a problem during acute management (appears following resolution of spinal shock and return of spinal cord reflexes).
6. Succinylcholine: safe for use during the first 24-48 hours.
7. Cervical spine fractures are missed on 5-15% of portable x-rays.
8. ABC's of lateral cervical spine x-rays
   A. C1-C7 and the C7-T1 junction must be visible.
   B. Alignment: anterior vertebral bodies, anterior and posterior spinal canal, and the spinous process tip.
   C. Bones: body, pedicle, lamina, spinous process.
   D. Cartilage: disc spaces and facet joints.
   E. Soft tissue space.

C. Extremity trauma

1. Fat emboli: associated with pelvic and long bone fractures; may cause pulmonary insufficiency, dysrhythmias, skin petechiae, and mental deterioration within first 3 days.

D. Burns

1. First-degree burns are limited to the epithelium, while second-degree burns extend into the dermis, and third-degree burns destroy the entire skin thickness. Major thermal burn; second degree burn involving at least 25% of body surface area or a third degree burn of at least 10% body surface area or all smoke inhalation injuries.
2. Indications for early intubation include hypoxemia not correctable with a face mask, upper airway edema, or presence of copious secretions.
3. Succinylcholine is contraindicated in burn patients (however, it may be used in the first 24 hours). Burn patients require higher than normal doses of nondepolarizing muscle relaxants.
4. Burn patients require large fluid resuscitation, up to 2-4 cc/kg per percent of body surface burned during the first 24 hours (with the first half given in the first 8 hours and the remainder over the next 16 hours).

E. Cardiac tamponade

1. Manifestations
   A. Dyspnea, orthopnea, tachycardia.
   B. Beck's triad: hypotension, distant heart sounds, distention of jugular veins.
   C. Paradoxical pulse (>10 mmHg decline in BP during inspiration).
   D. The principle hemodynamic feature is a decrease in cardiac output from a reduced stroke volume with an increase in central venous pressure. In the absence of severe left ventricular dysfunction, equalization of diastolic pressure occurs throughout the heart (RAP=RVEDP=PAEDP=LAP=LVEDP). Impairment of both diastolic filling and atrial emptying abolishes the y descent; the x descent is normal.
   E. EKG: ST segment changes, electrical alternans.
   F. CXR: silhouette normal or slightly enlarged.
   G. Echo: best diagnostic tool.
2. Anesthetic considerations
   A. Maintain filling pressures (to maximize stroke volume)
   B. Avoid bradycardia (tachycardia is a key compensatory mechanism for maintaining cardiac output).
   C. If compromised, support myocardial contractility with inotropic support.
   D. Avoid positive pressure ventilation until absolutely necessary or until the tamponade physiology has been relieved (increased intrathoracic pressure will impede venous return and exacerbate underfilling of the cardiac chambers).
   E. Pre-induction monitors: standard monitors plus arterial line and central venous line (and pulmonary artery

catheter if needed).

F. Hemodynamically unstable patients should receive pericardiocentesis (under local anesthesia) prior to induction (the removal of even a small amount of fluid can improve cardiac performance).

G. Induction: ketamine is the drug of choice, however, ketamine depresses myocardial contractility and may precipitate hemodynamic deterioration when used in the presence of hypovolemia and maximal sympathetic outflow.

6. Glasgow coma scale (Total Score Possible = 3-15):

A. Best motor response: 6-obeys commands; 5-localizes pain; 4-withdrawals; 3-flexion: decorticate rigidity; 2-extension: decerebrate rigidity; 1-no motor response.

B. Best verbal response: 5-oriented, conversant; 4-disoriented, conversant; 3-inappropriate words; 2-incomprehensible sounds; 1-no verbalization/response.

C. Eye opening: 4-spontaneous; 3-to verbal stimulation; 2-to pain; 1-no response.

7. Management of the trauma airway

A. Overview

1. Perform visual scan of patient for obvious injuries.
2. Obtain history from prehospital personnel and patient (if able).
3. Five rules of trauma airway management
   A. The stomach is always is full.
   B. The cervical spine is always unstable.
   C. Altered mental status equals head injury.
   D. Partial airway obstruction may progress rapidly to complete airway obstruction.
   E. The patient is always dry

B. Primary survey (ascertain ABCs) and resuscitation

1. Airway maintenance with cervical spine control
   A. Upon initial evaluation of the trauma patient, the airway should be assessed first to ascertain patency.
      1. Rapid assessment for signs of airway obstruction should include inspection for foreign bodies and facial, mandibular, or tracheal/laryngeal fractures that may result in airway obstruction.
      2. Talk to the patient, the ability to speak virtually guarantees a patent airway and confirms the patient's ability to breathe. However, the patients' status may deteriorate at any time, and therefor must be constantly reassessed.
      3. Measures to establish a patent airway should protect the cervical spine. The chin lift or jaw thrust maneuvers are recommended to achieve this task.
      4. Patients who arrive intubated, should have placement confirmed (i.e., bilateral breath sounds with good chest rise, direct laryngoscopy, or capnography).
      5. During your initial assessment, all patients should receive supplemental oxygen (face mask, bag-

valve mask, endotracheal tube, etc).

B. If needed, establish a definitive airway: orotracheal or nasotracheal intubation, jet insufflation of the airway, or surgical cricothyroidotomy. Emergent tracheostomy is rarely indicated.

C. Remember: assume a cervical spine injury in any patient with multi-system trauma, especially with an altered level of consciousness or a blunt injury above the clavicle. Maintain the cervical spine in a neutral position with inline stabilization when establishing an airway.

D. The potential for cervical spine injury makes airway management more complex in the trauma patient. A cervical spine injury should be suspected in all injury mechanisms involving blunt trauma. Patients with injury above the clavicles are at increased risk, and this is increased 4 fold if there is a clinically significant head injury (GCS < 9). Cervical spine injury is often occult, and secondary injury to the spinal cord must be avoided. Immobilization of the cervical spine must be instituted until a complete clinical and radiological evaluation has excluded injury.

2. Breathing and ventilation

A. Airway patency alone does not assure adequate ventilation. Ventilation involves adequate function of the lungs, chest wall, and diaphragm. Each component must be examined and evaluated rapidly.

B. Definitive control of the airway in patients who have compromised airways due to mechanical factors, who have ventilatory problems, or who are unconscious is achieved by endotracheal intubation.

3. Circulation with hemorrhage control

A. Hypotension following injury must be considered to be hypovolemic in origin until proved otherwise.

B. A minimum of two large-caliber intravenous catheters should be established.

C. All major trauma patients should have at least one level one transfuser connected to a large-caliber IV catheter.

C. Trauma intubations

1. Indications for airway intervention

A. Airway obstruction.

B. Hypoxia and hypercarbia (i.e., severe shock or cardiac arrest).

C. Controlled hyperventilation (patients with obvious intracranial injury or GCS of less than 9).

D. Protection against pulmonary aspiration (i.e, drug overdose patients).

E. Airway injury (e.g., GSW to the neck, inhalation injuries).

F. Sedation for diagnostic procedures (patients who are intoxicated or suffering from possible head injury that are unable to lie still for necessary diagnostic studies).

G. Prophylactic intubation (patients with impending respiratory failure or airway compromise).

H. Airway or midface injuries.
I. Large flail segment.

2. Preparation
   A. The urgency of airway intubation is the most important factor in planning which technique of securing the airway is the safest and most appropriate. One must evaluate and assess the risk of further cord injury given head and neck movement, the degree of cooperation from the patient, anatomy and trauma to the airway and one's own expertise in each technique.
   B. All multiple trauma patients should be assumed to have a cervical spine injury and a full stomach. Portable cervical spine x-rays miss 5% to 15% of injuries (cervical spines reported to be 'cleared' after a single lateral radiograph should still be treated as if an injury is present). Complete evaluation of the cervical spine may require a CT scan or multiple radiographs and clinical exam. Cervical spine injury is unlikely in alert patients without neck pain or tenderness.
   C. Patients who arrive ventilated with an esophageal obturator airway (EOA) should have a more definitive airway placed before the EOA is removed. After the tracheal has been intubated and placement confirmed, the stomach should be suctioned prior to the removal of the EOA.
   D. If possible, patients with potential spinal cord injuries, document any movement of extremities before and after intubation. On patients with possible head injuries, the trauma team may wish to do a preliminary neurological evaluation before the patient is induced and intubated.
   E. Airway assessment
      1. The airway should be examined to detect a potentially difficult intubation. Airway evaluation is typically done during the initial assessment. Remember, all multi-trauma patients are assumed to have a cervical spine injury and thus, have limited head and neck movement.
   F. Airway equipment (laryngoscope, endotracheal tubes, suction, etc) should be set-up prior to the patients arrival.
3. Endotracheal intubation
   A. Preoxygenation
      1. Preoxygenation helps prevent hypoxia during intubation. All patients should be preoxygenated.
      2. Administration of 100% oxygen to an individual with normal spontaneous ventilation for 3 minutes or 4-6 vital capacity breaths will generally result in 95%-98% nitrogen washout.
   B. Techniques
      1. The safest method of securing the trauma airway remains debatable. In general, the technique used should be the one the operator is most

familiar with. The ATLS manual recommends a nasotracheal tube in the spontaneously breathing patient, and orotracheal intubation in the apenic patient with manual in-line axial stabilization maintained throughout. The hard collar may interfere with intubation efforts and the front part may be removed to facilitate intubation as long as manual stabilization is in effect.

2. Oral/nasal airways: oral or nasal airways may help maintain a patent airway.
3. Orotracheal intubation: orotracheal intubation facilitated by the use of muscle relaxants and general anesthesia is the technique of choice for intubating the trachea of trauma patients (it is the fastest and surest method of intubating the trachea). Outcome data indicates that there is no difference in terms of incidence of neurological deficit between awake intubation (orotracheal or nasotracheal) and intubation under general anesthesia as long as in-line cervical stabilization was used.
4. Nasotracheal intubation
    A. Following the introduction of muscle relaxants and rapid induction methods, this technique has decreased in popularity.
    B. Contraindications to nasotracheal intubation include: apnea (relative); upper airway foreign body, abscess, or tumor; nasal obstruction; central facial fractures; acute epiglottitis (blind technique); basal skull fractures; coagulopathy; and cardiac or other prosthesis (relative).
5. Other methods of tracheal intubation
    A. Many other methods of tracheal intubation exist. The bottom line is do what you do best but do it carefully and safely.
    B. Awake intubation is also a feasible option and is favored by some practitioners. It has been shown to be safe in the patient with cervical spine injury. It may be performed via the nasotracheal route, direct oral laryngoscopy or by fiberoptic technique. Successful fiberoptic tracheal intubation requires a cooperative patient, a secretion and blood free airway, a pharynx unrestricted by edema and adequate supraglottic and infraglottic anaesthesia. Such ideal conditions often do not exist, and local anaesthetic preparation of the airway is time consuming and might increase the risk of aspiration.
    C. See below for other methods.

C. Rapid sequence with cricoid pressure.

1. Technique: One person removes the c-collar and holds "in-line cervical stabilization" (usually surgery resident), another person applies cricoid pressure and pushes drugs, while the third person performs the intubation. After intubation, replace the c-collar. Maintain in-line cervical stabilization until the c-collar is replaced.
2. Commonly used drugs (the ideal induction agent probably does not exist. The agent of choice should be based of the individuals experience).
   A. Thiopental.
   B. Etomidate.
   C. Ketamine.
   D. Lidocaine.
   E. Succinylcholine.
3. Bottom line: whether or not drugs are used depends on severity of the injury. Reduced doses of induction agent or no induction agent are at times appropriate in severely injured, obtunded patients.

D. The difficult trauma airway

1. If there is difficulty or delay in intubating the trachea in any trauma patient with respiratory comprise, a tracheotomy or cricothyroidotomy should be performed immediately. In patients who appear to have a difficult airway, surgery may want to start preparing for a surgical airway while you attempt to intubate the patient.
2. Cricothyroidotomy: the need for cricothyroidotomy due to severe maxillofacial trauma or an inability to perform oral-tracheal intubation occurs in less than 1% of all trauma patients requiring intubation on admission. It may be used as a primary airway, with injuries to the pharynx for example, or after failure of orotracheal intubation. It may be a full surgical approach or via a percutaneous needle cricothyroidotomy with high flow oxygen.
3. Laryngeal mask airway (LMA): the LMA is gaining wider support in the management of patients with cervical spine injuries. As well as maintaining the airway, a tracheal tube (size 6 or less) may be placed, either blindly or via flexible fiberoptic laryngoscopy. The LMA does not however protect the airway from aspiration, and by acting as a bolus in the pharynx, may actually relax the lower oesophageal sphincter and increase reflux. It's use should probably be limited to maintenance of the airway after a failed attempt at intubation.
4. Combitube: the Combitube is a double lumen tube inserted blindly into the oesophagus or trachea

The position of the tube is confirmed by the presence of breath sounds or capnography. By inflating one of the two cuffs present, the lungs may then be ventilated. Problems arise after positioning with definitive securing of a tracheal tube, and again protection of the airway from aspiration, although stomach suctioning is possible through the gastric port.

E. Confirm endotracheal tube placement

1. As with all intubations, confirmation of tracheal intubation should be done. The emergency department trauma rooms are equipped with end-tidal CO_2 monitoring capability.

Anesthesia for Ophthalmologic Surgery

1. Physiology of intraocular pressure

A. Normal intraocular pressure is maintained between 10 and 20 mmHg.

B. Aqueous humor is formed at a rate of 2 microliters/min. Two thirds is secreted by the ciliary body via an active sodium-pump mechanism. One third is produced by passive ultrafiltration through vessels on the anterior iris. Fluid ultimately drains through the Canal of Schlemm.

C. Intraocular pressure is controlled primarily by regulation of the outflow resistance at the trabecular meshwork. Acute changes in choroidal blood volume can produce rapid increases in intraocular pressure. Hypercapnia can lead to choroidal congestion and increased intraocular pressure. The increases in venous pressure associated with coughing, straining, or vomiting can raise IOP to 30 to 40 mmHg. Similar increases can be seen at intubation. Intraocular pressure can also be increased by extrinsic compression of the globe. The force of the eyelid in a normal blink may cause an increase of 10 mmHg; a forceful lid squeeze can increase IOP to over 50 mmHg A poorly placed anesthesia mask could increase IOP to the point of zero blood flow.

D. Choroidal blood flow is normally autoregulated.

E. Glaucoma

1. In open-angle glaucoma, sclerosis of the trabecular meshwork is believed to impede aqueous drainage.
2. Closed-angle glaucoma occurs when the peripheral iris swells or is displaced anteriorly and closes the anterior chamber angle to cause an obstruction to aqueous drainage.

2. Oculocardiac reflex

A. Traction on extraocular muscles or pressure on the globe can elicit cardiac dysrhythmias ranging from bradycardia and ventricular ectopy to sinus arrest or ventricular fibrillation.

B. The reflex is trigeminovagal. The afferent limb is from orbital contents via the long and short ciliary nerves to the ciliary ganglion to the ophthalmic division of the trigeminal nerve to the gasserian ganglion to the sensory nucleus of the trigeminal nerve near the fourth ventricle. Internuncial fibers travel in the reticular formation to the motor nucleus of the vagus nerve. From there efferents travel via the cardiac depressor nerves of the vagus to the heart. The reflex fatigues with repeated traction on the extraocular muscles.

C. Prevention

1. Retrobulbar block is not uniformly effective in preventing the reflex (retrobulbar block may elicit the oculocardiac reflex).
2. Anticholinergic medication can be effective, however caution must be used in the elderly.
3. Deep anesthesia.
4. Factors associated with increased susceptibility to the development of ocularcardiac reflex are anxiety, hypoxia, hypercarbia, and light anesthesia.

D. Treatment
   1. Request the surgeon to stop manipulation.
   2. Assess adequate ventilation, oxygenation, and depth of anesthesia.
   3. If severe or persistent bradycardia, give atropine (7-10 mcg/kg).
   4. In recurrent episodes, infiltration of the rectus muscles with local anesthetics.

3. Intraocular gas expansion

A. A gas bubble may be injected into the posterior chamber during vitreous surgery to flatten a detached retina.

B. The air bubble is absorbed within 5 days by gradual diffusion.

C. Sulfur hexafluoride, an inert gas that is less soluble in blood than nitrogen, provides a longer duration (up to 10 days) in comparison with an air bubble.

D. Nitrous oxide should be discontinued at least 15 minutes prior to the injection of air or sulfur hexafluoride. Nitrous oxide should be avoided until the bubble is absorbed (5 days for air and 10 days for sulfur hexafluoride injection).

4. Anesthetic drugs

A. Most anesthetic drugs either lower or have no effect on intraocular pressure. An exception is ketamine, and possibly etomidate.
   1. Ketamine effects are controversial, but is generally felt to moderately increase intraocular pressure. Ketamine increases choroidal blood flow, increases nystagmus, and increases extraocular muscle tone via blepharospasm.
   2. Etomidate, which is associated with a high incidence of myoclonus (10-60%), may increase intraocular pressure.

B. Succinylcholine can cause a 5-10 mmHg increase in intraocular pressure for 5-10 minutes. Succinylcholine can potentially increase intraocular pressure by dilating choroidal blood vessels and increases in extraocular muscle tone. Pretreatment with a defasiculating dose of a nondepolarizing muscle relaxant does not reliably eliminate the effect of succinylcholine on intraocular pressure. Nondepolarizing muscle relaxants do not increase intraocular pressure.

5. Systemic effects of ophthalmic drugs

A. Anticholinesterases (echothiophate, phospholine iodide): systemic absorption leads to inhibition of plasma cholinesterase which may lead to prolongation of succinylcholine's duration of action. Takes 3 weeks for pseudocholinesterase levels to return to 50% of normal. The metabolism of mivacurium and ester-type local anesthetics may also be affected.

B. Cholinergics (pilocarpine, acetylcholine): used to induce miosis; toxicity may manifest in bradycardia or acute bronchospasm.

C. Anticholinergics (atropine, scopolamine): used to cause mydriasis; systemic absorption may lead to tachycardia, dry skin, fever, and agitation.

D. Beta-blockers (timolol maleate): systemic absorption may cause beta-blockade (bradycardia, bronchospasm, or exacerbation of congestive heart failure). Betaxolol seems to be oculo-specific with minimal side effects.

E. Carbonic anhydrase inhibitors (acetazolamide, diamox): used to decrease aqueous production; induces an alkaline diuresis. Side

effects include diuresis and hypokalemic metabolic acidosis.

6. Retrobulbar blockade

A. Technique: local anesthetic is injected behind the eye into the cone formed by the extraocular muscles. Lidocaine and bupivacaine are the most commonly used local anesthetics. Hyaluronidase, a hydrolyzer of connective tissue polysaccharides, is commonly added to enhance the spread of local anesthetic.

B. Complications: retrobulbar hemorrhage, globe perforation, optic nerve atrophy, convulsions, oculocardiac reflex, loss of consciousness, and respiratory arrest.

C. Post-retrobulbar apnea syndrome: due to injection of local anesthetic into the optic nerve sheath with spread into the cerebrospinal fluid. Apnea typically occurs within 20 minutes and may last 15-60 minutes. Adequacy of ventilation must be constantly monitored in patients with retrobulbar blocks.

7. Facial nerve block

A. Facial nerve block prevents squinting of the eyelids.

B. Major complications of this block is subcutaneous hemorrhage.

Anesthesia for Special Cases

Anesthesia for Transurethral Resection of the Prostate (TURP)

1. Complications

A. Intravascular absorption of irrigating fluid: The amount of solution absorbed depends primarily on three factors: (1) the hydrostatic pressure of the irrigating fluid, (2) the duration of time sinuses are exposed to irrigating fluid (10 to 30 ml of irrigating fluid is absorbed per minute of irrigation time), and (3) the number and sizes of the venous sinuses opened during resection. Absorption of the irrigating fluid can result in the following: fluid overload, serum hypoosmolality, hyponatremia, hyperglycemia, hyperammonemia, hemolysis.

B. Autotransfusion secondary to lithotomy position.

C. Hypothermia.

D. Bacteremia: 10% incidence in patients with sterile urine and 50% incidence in patients with infected urine.

E. Blood loss: related to vascularity of the prostate gland, the surgeon's experience and technique, weight of the prostate resected (approximately 15 ml/gm of resected tissue), length of the operation.

F. Perforation of bladder or urethra with extravasation, either extraperitoneal or intraperitoneal.

G. Transient blindness: attributed to absorption of glycine and its metabolic byproduct, ammonia, acting as an inhibitory neurotransmitter in the retina.

H. CNS toxicity: result of oxidative biotransformation of glycine to ammonia.

I. CNS symptoms, including apprehension, irritability, confusion, headache, seizures, transient blindness, and coma, have all been attributed to hyponatremia and hypoosmolarity.

2. Management of TURP Syndrome and hyponatremia

A. Obtain immediate serum sodium and arterial blood gas.

B. Serum sodium >120 mEq/L: obtain hemostasis; terminate transurethral surgery; oxygen with mask or nasal cannula; fluid restriction; brisk diuresis with or without loop diuretics.

C. Serum sodium <120 mEq/L: obtain hemostasis; terminate transurethral surgery; oxygen with mask or nasal cannula; fluid restriction; loop diuretics; consider hypertonic saline (e.g., 3% or 5% saline) infused at a rate which does not exceed 100 ml/hr; allow sodium to rise by 0.5-2.0 mEq/L/hr; stop hypertonic saline and loop diuretics once sodium ranges 120 to 130 mEq/L.

Anesthesia for Electroconvulsive Therapy (ECT)

1. Cardiovascular response

A. Initial parasympathetic outflow, immediately after stimulation, that may result in bradycardia.

B. The parasympathetic response is followed by a sympathetic outflow, which produces hypertension and tachycardia that usually lasts 5-10 minutes.

2. Anesthetic Management

A. Methohexital 0.5-1.0 mg/kg and succinylcholine 0.25-0.5 mg/kg.

B. Place blood pressure cuff on the opposite arm of the IV and inflate prior to the administration of succinylcholine to allow for motor expression of the seizure.

C. Induced seizures should last greater than 25 seconds and should be terminated if longer than 3 minutes.

Myasthenia Gravis

1. Myasthenia gravis is characterized by weakness and easy fatigability of skeletal muscle. The weakness is thought to be due to autoimmune destruction or inactivation of postsynaptic acetylcholine receptors at the neuromuscular junction. The course of the disease is marked by exacerbations and remissions. Muscle strength characteristically improves with rest but deteriorates rapidly with repeated effort.

2. Osserman classification

A. Type I: Involvement of extraocular muscles only.

B. Type IIa: Mild skeletal muscle weakness, spares muscles of respiration.

C. Type IIb: More severe skeletal muscle weakness with bulbar involvement.

D. Type III: Acute onset. rapid deterioration, severe bulbar and skeletal muscle involvement.

E. Type IV: Late, severe involvement of bulbar and skeletal muscle.

3. Treatment

A. Treatment is with anticholinesterase drugs, immunosuppressants, glucocorticoids, plasmapheresis, and thymectomy.

B. Anticholinesterase drugs (most commonly pyridostigmine) inhibiting the breakdown of acetylcholine by tissue cholinesterase, increasing the amount of acetylcholine at the neuromuscular junction.

C. Cholinergic crisis: characterized by increased weakness and excessive muscarinic effect, including salivation, diarrhea, miosis, and bradycardia.

D. Edrophonium test: used to differentiate a cholinergic form a myasthenic crisis. Increased weakness after up to 10 mg of intravenous edrophonium is indicative of cholinergic crisis, whereas increasing strength implies myasthenic crisis.

4. Pre-op predictors for post-op ventilation (after transternal thymectomy)

A. Duration of disease greater than 6 years.

B. Presence of COPD or other lung disease unrelated to myasthenia.

C. Pyridostigmine dose greater than 750 mg/day.

D. Preoperative FVC less than 2.9 liters.

5. Anesthetic concerns

A. Muscle relaxants should generally be avoided. The response to succinylcholine is unpredictable. Patients may manifest a relative resistance, a prolonged effect, or an unusual response (phase II block).

Myasthenic Syndrome

1. Myasthenic syndrome, also called **Eaton-Lambert syndrome**, is a paraneoplastic syndrome characterized by proximal muscle weakness that typically affects the lower extremities. Myasthenic syndrome is usually associated with small-cell carcinoma of the lung. In contrast to myasthenia gravis, the muscle weakness improves with repeated effort and is unaffected by anticholinesterase drugs.
2. Patients with the myasthenic syndrome are very sensitive to both depolarizing and nondepolarizing muscle relaxants. The response to other drugs used in anesthesia is usually normal.

Anesthesia for Organ Harvest

1. The donor

A. Brain death should be pronounced prior to transfer of the donor to the OR.

B. Exclusion of other causes simulating brain death
   1. Body temperature less than 95 degrees F.
   2. Absence of drug intoxication or neuromuscular blocking agents.
   3. Corrected metabolic abnormalities.

C. Clinical criteria
   1. Cerebral unresponsiveness, irreversible coma.
   2. Brain stem unresponsiveness.
      A. Fixed and dilated pupils, doll's eyes, negative caloric test, absent corneal reflex.
      B. Absent gag and cough reflex, apnea (no respiratory efforts of the ventilator with $PaCO_2$ greater than 60 mmHg).
   3. No posturing (spinal reflexes may be present).

D. Ancillary tests
   1. Isoelectric electroencephalogram.
   2. Absent cerebral blood flow by intracranial angiography or nuclear brain scan.

2. Donor management

A. Overall goals are restoration and maintenance of hemodynamic and vascular stability. Hemodynamics should be maintained as followed (for adults):
   1. Systolic blood pressure greater than 100 mmHg.
   2. Central venous pressure 10-12 mmHg.
   3. Urine output greater than 100 cc/hour.
   4. P_aO_2 greater than 100 mmHg.

B. Brain death induces many physiologic abnormalities.
   1. Cardiovascular instability is a common feature, secondary to loss of neurologic control of the myocardium and vascular tree. Most transplant teams prefer fluid resuscitation to vasoconstrictors. Keep systolic blood pressure greater than 100 mmHg and mean arterial pressure greater than 70 mmHg
   2. Central diabetes insipidus may occur from hypothalamic failure resulting in extreme salt and water wasting from the kidneys. Treatment is important to reverse the massive loss of fluid and electrolytes that may occur. Aqueous Pitressin should be administered in doses of 10 units intravenously every 4 hours to bring urine output down to 150-200 cc per hour.

3. Loss of thermoregulatory control. Most patients are unable to control their body temperatures and have a steady downward drift in core.
4. Neurogenic pulmonary edema may be present.
5. Coagulopathy: the release of large amounts of tissue fibrinolytic agent from a necrotic brain probably initiates coagulopathy.

C. Hypoxia: pulmonary insufficiency secondary to trauma and/or shock should be treated with mechanical ventilation, positive end-expiratory pressure (PEEP), and inspired oxygen fraction sufficient to maintain adequate peripheral oxygen delivery. Potential heart-lung donors should receive the lowest inspired oxygen possible to minimize pulmonary oxygen toxicity.

D. Overall hypovolemia is the most important variable affecting donor organ perfusion. Ringer's lactate should be infused to establish a central venous pressure of 10-12 mmHg. Hematocrit should be maintained about 30%.

E. Anesthesia per se is not needed in the brain dead patient. However, significant hemodynamic responses to surgical stimuli commonly occur in the brain-dead donor during organ harvesting. These responses may reflect some residual lower medullary function (visceral and somatic reflexes), but they do not invalidate the diagnosis of brain death (cortical and brain-stem function are absent). Movement secondary to spinal reflex action should be controlled. Patients are routinely declared dead prior to going to the operating room.

3. Organ preservation

A. Organ preservation is achieved in two ways: (1) by decreasing metabolic demand and (2) by increasing the supply of vital substances.

B. Metabolic demand is decreased by using hypothermia and/or pharmacologic inhibition. For each reduction of 10 degrees C, metabolic rate is decreased by a factor of 2-3. Most centers today use a combination of topical hypothermia and cold, hyperkalemic cardioplegia to achieve donor heart protection.

C. Most cardiac transplant teams prefer to limit the ischemia time of the donor heart to four hours or less. Ischemia time begins when the aortic cross-clamp is placed during the harvest and ends when the aortic clamp is released in the recipient.

Anesthesia for Laparoscopic Surgery

1. **Advantages:** benefits include decreased postoperative pain, less postoperative pulmonary impairment, a reduction in postoperative ileus, shorter hospital stays, earlier ambulation, and smaller surgical scars.
2. **Pulmonary effects:** hallmark of laparoscopy is the creation of a pneumoperitoneum with pressurized CO_2 (up to a pressure of 30 cm H_2O). The resulting increase in intra-abdominal pressure displaces the diaphragm cephalad, causing a decrease in lung compliance and an increase in peak inspiratory pressure. Atelectasis, diminished functional residual capacity, ventilation/perfusion mismatch, and pulmonary shunting contribute to a decrease in arterial oxygenation. The high solubility of CO_2 increases systemic absorption which can lead to increased arterial CO_2 levels and decreased pH.
3. **Patient position**: trendelenburg is often associated with a decrease in FRC, VC, TLV, and pulmonary compliance.
4. **Cardiac effects:** moderate insufflation can increase effective cardiac filling because blood tends to be forced out of the abdomen and into the chest. Higher insufflation pressures (greater than 25 cm H_2O), however, tends to collapse the major abdominal veins which compromises venous return and leads to a drop in preload and cardiac output in some patients. Hypercarbia, if allowed to develop, may stimulate the sympathetic nervous system and thus increase blood pressure, heart rate, and risk of dysrhythmias.
5. **Anesthetic technique:** local, regional and general anesthesia have all been used, however, general anesthesia with endotracheal intubation is the preferred technique.
6. **Complications:** hemorrhage, peritonitis, subcutaneous emphysema,s pneumomediastinum, or pneumothorax, and venous air embolism. Vagal stimulation during trocar insertion, peritoneal insufflation, or manipulation of viscera can result in bradycardia and even sinus arrest.

Postanesthesia Care Unit (PACU)

1. **Hypotension:** arterial hypoxemia; hypovolemia (most common); spurious (cuff too wide, transducer not calibrated); pulmonary edema (excess fluids); cardiac dysrhythmias; decreased systemic vascular resistance (sepsis); pneumothorax; cardiac tamponade.
2. **Hypertension:** arterial hypoxemia; hypothermia; spurious (cuff too narrow, transducer not calibrated or overshoot); pre-existing essential hypertension; enhanced sympathetic nervous system activity (pain, carinal stimulation, bladder distention); excess fluid administration.
3. **Laryngospasm**
   A. Laryngospasm is a forceful involuntary spasm of the laryngeal musculature caused by sensory stimulation of the superior laryngeal nerve. Triggering stimuli include pharyngeal secretions or extubating in stage 2.
   B. Treatment: initial treatment includes 100% oxygen, anterior mandibular displacement, and gentle CPAP (may be applied by face mask). If laryngospasm persists and hypoxia develops, succinylcholine (0.25-1.0 mg/kg; 10-20 mg for average adult) should be given in order to paralyze the laryngeal muscles and allow controlled ventilation.
   C. The large negative intrathoracic pressures generated by the struggling patient in laryngospasm can result in development of pulmonary edema even in healthy young adults.
4. **Treatment of glottic edema and subglottic edema**
   A. Administer warm, humidified oxygen by mask.
   B. Inhalation of racemic epinephrine 2.25% (0.5-1 ml in 2 ml NS); repeated every 20 min.
   C. Dexamethasone 0.1-0.5 mg/kg IV.
   D. Possible reintubation with smaller tube.
5. **Arterial hypoxemia:** right to left shunt (atelectasis); ventilation to perfusion mismatch; decreased cardiac output; aspiration; alveolar hypoventilation (residual effects of drugs); pulmonary embolus; pulmonary edema; pneumothorax; posthyperventilation hypoxia; increased oxygen consumption (shivering); elderly; obese.
6. **Cardiac dysrhythmias:** arterial hypoxemia; hypovolemia; pain; hypothermia; anticholinesterases; myocardial ischemia; electrolyte abnormalities; respiratory acidosis; hypertension; digitalis intoxication.
7. **Agitation**
   A. Serious systemic disturbances (hypoxia, acidosis or hypotension) or bladder distention should be ruled out.
   B. After the above disturbances and pain have been excluded, persistent agitation can be treated with midazolam, 1-2 mg IV (0.05 mg/kg in children).
8. **Shivering**
   A. Shivering can occur secondary to hypothermia or the effects of anesthetic agents (most often volatile anesthetics).
   B. Shivering should be treated with warming measures. Small doses of meperidine (12.5-25 mg) IV and can dramatically reduce shivering.

9. Persistent obtundation

A. Check preoperative level of consciousness, residual anesthetic (may reverse with narcan, flumazenil or physostigmine), recurarization (check twitch monitor), severe hypothermia (<33°C), hypoglycemia, neurologic (intraoperative stroke, paradoxical air embolism, hypotension/hypertension, cerebral bleed).

10. Nausea/vomiting

A. Risk factors
   1. Patient factors: younger age, female, obesity, anxiety, gastroparesis, history of postoperative nausea/vomiting or motion sickness.
   2. Surgical procedures: gynecological, abdominal, ENT, ophthalmic, plastic surgery.
   3. Anesthetic factors: premedicants (morphine and other opioid compounds), anesthetics agents (nitrous oxide, inhalational agents, etomidate, methohexital, balanced techniques), anticholinesterase reversal agents, gastric distention, longer duration of anesthesia.
   4. Postoperative factors: pain, dizziness, movement after surgery, premature oral intake, opioid administration.

B. Treatment
   1. Droperidol 25-50 mcg/kg.
   2. Metoclopramide 0.15 mg/kg or 10 mg (for adults).
   3. Ondansetron 0.05-0.15 mg/kg**.**

11. Pain control

A. Meperidine 10-20 mg (0.25-0.5 mg/kg in children).

B. Morphine 2-4 mg (0.025-0.05 mg/kg in children).

12. Discharge criteria

A. Stable vital signs, alert and oriented (or to baseline), able to maintain adequate oxygen saturation, free of nausea/vomiting, absence of bleeding, adequate urine output, adequate pain control, stabilization or resolution of any problems, movement of extremity following regional anesthesia.

B. Aldrete scoring system (a score of 10 indicates the best possible condition for discharge from the PACU)
   1. Activity (able to move voluntarily or on command)
      A. 4 extremities: 2
      B. 2 extremities: 1
      C. 0 extremities: 0
   2. Respiration
      A. Able to deep breath and cough freely: 2
      B. Dyspnea, shallow or limited breathing: 1
      C. Apneic: 0
   3. Circulation
      A. BP ± 20 mm of preanesthesia level: 2
      B. BP ± 20 to 50 mm pr preanesthesia level: 1
      C. BP ± 50 mm of preanesthesia level: 0
   4. Consciousness
      A. Fully awake: 2
      B. Arousable on calling: 1
      C. Not responding: 0
   5. Color
      A. Normal: 2

   B. Pale, dusky, blotchy: 1
   C. Cyanotic: 0

C. Postanesthesia discharge scoring system (the total score is 10; patients scoring 9 or greater are considered fit for discharge)
   1. Vital signs
      A. Within 20% of preoperative value: 2
      B. Within 20-40% of preoperative value: 1
      C. Within 40% of preoperative value: 0
   2. Ambulation and mental status
      A. Oriented x 3 AND has a steady gait: 2
      B. Oriented x 3 OR has a steady gait: 1
      C. Neither: 0
   3. Pain or nausea/vomiting
      A. Minimal: 2
      B. Moderate: 1
      C. Severe: 0
   4. Surgical bleeding
      A. Minimal: 2
      B. Moderate: 1
      C. Severe: 0
   5. Intake and output
      A. Has had PO fluids AND voided: 2
      B. Has had PO fluids OR voided: 1
      C. Neither: 0

Malignant Hyperthermia

1. **Definition:** a fulminant skeletal muscle hypermetabolic crisis triggered by anesthetic agents. Etiology multiple, however may be genetic defect in some MH families. The gene for malignant hyperthermia is also the genetic coding site for the calcium release channel of skeletal muscle sarcoplasmic reticulum (ryanodine receptor). High incidence in patients (especially children) with masseter muscle rigidity.
2. **Clinical findings (variable)**
   A. Signs of onset: tachycardia, tachypnea, hypercarbia (increased end-tidal CO_2 most sensitive clinical sign).
   B. Early: sometimes muscle rigidity (including isolated masseter spasm) following succinylcholine, tachycardia, tachypnea, unstable blood pressure, arrhythmias, cyanosis, mottling, sweating, rapid temperature increase, and cola-colored urine.
   C. Late (6-24 hours): pyrexia, skeletal muscle swelling, left heart failure, renal failure, DIC, hepatic failure.
   D. Laboratory: respiratory and metabolic acidosis, hypoxemia, increased serum levels of K, Ca, myoglobin, CPK, and myoglobinuria.
3. **MH triggering agents:** succinylcholine and all the volatile agents (halothane most potent).
4. **Drugs that do not trigger MH:** barbiturates, opioids, benzodiazepines, propofol, etomidate, nitrous oxide, local anesthetics, nondepolarizing muscle relaxants.
5. **Monitoring after presumed MH:** routine monitors plus arterial line, CVP (possible PA cath), and foley.
6. **Incidence**
   A. Children: approx 1:12,000 general anesthetics.
   B. Adults: approx 1:40,000 general anesthetics when succinylcholine is used; approx 1:220,000 general anesthetics when succinylcholine is not used.
   C. Familial autosomal dominant transmission with variable penetrance.
7. **Mortality:** 10% overall; up to 70% without dantrolene therapy; with early therapy less than 5%.
8. **Management of patient with masseter muscle rigidity**
   A. If the response to succinylcholine is difficulty in opening the jaw ("difficult intubation"), then continue with nontriggering agents, monitor carefully, obtain postop CPK's q 6 hours x 4, follow urine volume and myoglobin, and keep in recovery room for four hours. If postop CPKs go above 20,000 units per liter, assume (>95%) that the patient is MHS.
   B. If the response to succinylcholine is such that the "jaw could not be opened," then stop the anesthetic immediately, monitor carefully, obtain postop CPKs, monitor urine volume and myoglobin, and keep in the recovery room for four hours.
9. **Prophylaxis**
   A. Dantrolene: dantrolene pretreatment is not necessary in all cases. When used, the recommended pretreatment dose is 2.5 mg/kg IV within 30 minutes of anesthetic induction. Use dantrolene with extreme caution in patients with concurrent myopathy.
   B. Clean anesthesia machine and delivery circuit. Ten liters per minute

of oxygen flow through the circuit for 20 minutes will effectively purge the machine of residual. Changing the fresh gas hose will hasten the reduction of the concentration of inhalation agents. Fresh carbon dioxide absorbent and fresh delivery tubing are recommended.

10. Malignant hyperthermia treatment protocol

A. **STOP TRIGGERING ANESTHETIC AGENTS IMMEDIATELY**, conclude surgery as soon as possible. Call for help. Continue with safe agents if surgery cannot be stopped.

B. Hyperventilate: 100% oxygen at high flows. Use new circuit and soda lime.

C. Administer: dantrolene 2.5 mg/kg IV. Continue until all signs normalize (up to 10 mg/kg).

D. Correct metabolic acidosis: administer sodium bicarbonate, 1-2 mEq/kg IV guided by arterial pH and pCO_2. Follow with ABG.

E. Hyperkalemia: correct with bicarbonate or with glucose, 25-50 gms IV, and regular insulin, 10-20 u.

F. Actively cool patient:
   1. Iced IV NS (not ringer's lactate) 15 ml/kg every 10 minutes times three if needed. Monitor.
   2. Lavage stomach, bladder, rectum, peritoneal and thoracic cavities with iced NS.
   3. Surface cool with ice and hypothermia blanket.

G. Maintain: UOP > 1-2 ml/kg/hr. If needed, mannitol 0.25 g/kg IV, furosemide 1 mg/kg IV (up to 4 times).

H. Labs: UOP, PT, PTT, platelets, urine myoglobin, ABG, K, Ca, lactate, CPK.

I. Treat persistent ventricular arrhythmias with procainamide, 3 mg/kg IV (max 15 mg/kg).

J. Consider invasive monitoring of arterial blood pressure and central venous pressure.

K. Postoperatively: continue dantrolene 1 mg/kg IV every 6 hours x 72 hrs to prevent recurrence. Observe in ICU until stable for 24-48 hrs.

L. Calcium channel blockers should not be given when dantrolene is administered as hyperkalemia and myocardial depression may occur.

M. If necessary, consult on-call physicians: MH Alert Hotline (209) 634-4917: ask for Index Zero. Report MH events to The North American MH Registry: (717) 531-6936.

Allergic Drug Reactions

Anaphylaxis

1. Clinical manifestations of anaphylaxis

- **A. Cardiovascular:** hypotension, tachycardia, dysrhythmias.
- **B. Pulmonary:** bronchospasm, cough, dyspnea, pulmonary edema, laryngeal edema, hypoxemia.
- **C. Dermatologic:** urticaria, facial edema, pruritus.

2. Treatment of anaphylactic and anaphylactoid reactions

- **A. Initial therapy**
  1. Discontinue drug administration and all anesthetic agents.
  2. Administer 100% oxygen.
  3. Intravenous fluids (1-5 liters of LR).
  4. Epinephrine (4-8 mcg IV bolus with hypotension, titrate as needed; 0.1-0.5 mg IV with cardiovascular collapse).
- **B. Secondary treatment**
  1. Antihistamines (benadryl 0.5-1 mg/kg or 50-75 mg IV).
  2. Catecholamine infusions (starting doses: epinephrine 2-4. mcg/min, norepinephrine 2-4 mcg/min, or isoproterenol 0.5-1 mcg/min as a drip, titrated to effect).
  3. Aminophylline (5-6 mg/kg IV over 20 minutes with persistent bronchospasm).
  4. Corticosteroids (0.25-1 gram hydrocortisone; alternately 1-2 grams methylprednisolone).
  5. Sodium bicarbonate (0.5-1 mEq/kg with persistent hypotension or acidosis).
  6. Airway evaluation (prior to extubation).

Venous Air Embolism

1. Background information

A. Air can be entrained into a vein whenever there is an open vein and a negative intravenous pressure relative to atmospheric pressure. Generally, this can occur any time the surgical field is above right atrial level (neurosurgical procedures and operations involving the neck, thorax, abdomen, pelvis, open heart, liver and vena cava laceration repairs, total hip replacement, and vaginal delivery associated with placenta previa.

B. Incidence is highest during sitting craniotomies (20-40%).

C. Paradoxic air emboli are more likely to occur in patients with probe-patent foramen ovale.

2. Diagnosis

A. Transesophageal two-dimensional echocardiography (TEE): thought to be the most sensitive. Sensitivity = 0.015 ml of air/kg/min. While the TEE has the added benefits of detecting the size of the bubbles and transatrial passage, it is hard to use and not that specific.

B. Doppler: sensitivity equals 0.02 ml of air/kg/min.

C. Decreased PaO_2, TcO_2, and increased ETN_2 (most specific) normally occur before one sees a sudden decrease in $ETCO_2$ and/or an increase in CVP.

D. During controlled ventilation of the lungs, sudden attempts by the patient to initiate a spontaneous breath (gasp reflex) may be the preceding indication of venous air embolism.

E. Hypotension, tachycardia, cardiac dysrhythmias and cyanosis are late signs of a venous air embolism.

F. Consequences: depend of the volume and rate of air entry:

1. CVP increases 0.4 ml of air/kg/min.
2. Heart rate increases at 0.42 ml of air/kg/min.
3. EKG changes occur at 0.6 ml of air/kg/min.
4. Blood pressure decreases at 0.69 ml of air/kg/min.
5. Mill wheel murmur is heard at 2.0 ml of air/kg/min.
6. Anything much greater than 2.0 ml of air/kg/min is potentially lethal.

3. Treatment

A. Notify surgeon to flood surgical field with saline or pack and apply bone wax to the skull edges until the entry site identified.

B. Nitrous oxide should be discontinued and 100% oxygen given.

C. The central venous catheter should be aspirated in an attempt to retrieve the entrained air.

D. Give intravascular volume infusion to increase CVP.

E. Support cardiovascular system with volume, inotropes, and/or vasopressors.

F. Increase venous pressure with bilateral jugular vein compression, may slow air entrainment and help the surgeon identify the source of the embolus.

G. Consider PEEP in an effort to increase cerebral venous pressure; however, reversal of the normal transatrial pressure gradient may promote paradoxical embolism.
H. If not identified continue with placing the patient in the left lateral decubitus position with a slight head-down tilt in an attempt to dislodge a possible air lock.

Latex Allergy

1. The severity of allergic reactions to latex-containing products ranges from mild contact dermatitis to life threatening anaphylaxis.
2. **Risk factors for latex allergy**
   A. Chronic exposure to latex and a history of atopy increases the risk of sensitization.
   B. Patients undergoing frequent procedures with latex items (eg, repeated urinary bladder catheterization).
   C. Patients with neural tube defects (meningomyelocele, spina bifida).
   D. Congenital abnormalities of the genitourinary tract.
3. **Pathophysiology**
   A. Most reactions involve a direct IgE-mediated immune response to polypeptides in natural latex.
   B. Some cases of contact dermatitis may be due to a type 4 sensitivity reaction to chemicals introduced in the manufacturing process.
4. **Preoperative evaluation/workup**
   A. History: take a careful history in patients at risk, particularly those with co-existing atopy and/or multiple allergies. Ask for a history of balloon or glove intolerance and allergies to medical products used in chronic care (e.g. catheters). Elective patients in whom you suspect latex allergy should be referred to an allergist.
   B. Diagnostic tests: routine diagnostic testing in the at-risk population is not recommended (only those with a positive history). Test available include
      1. Skin-prick test: less sensitive than intradermal test but more sensitive than RAST.
      2. Radioallergosorbent test (RAST): an in-vitro test for IgE antibodies in the patient's serum.
   C. Pre-operative medications: routine preoperative H_1 and H_2 blockers and steroids are no longer recommended.
   D. Scheduling: since latex is an aeroallergen and present in the operating room air for at least an hour after the use of latex gloves, whenever possible your patient should be scheduled as the first case of the day.
5. **Anesthesia equipment**
   A. Common anesthesia equipment that contains latex include gloves, tourniquets, endotracheal tube, ventilator bellows, intravenous injection ports, blood pressure cuffs, and face masks. Medical products containing latex are required to label their products accordingly.
   B. Non-latex supplies that are commonly required include glass syringes, drugs in glass in ampules, IV tubing without latex injection ports, neoprene gloves, ambu bag with silicone valves, and neoprene bellows for the Ohmeda ventilator.
   C. The most important precaution is the use of non-latex gloves.
   D. Misc equipment: sleeve on the fibreoptic bronchoscope is non-latex, esophageal stethoscope is non-latex, cuff on the LMA airway is non-latex (silicon).

6. Preoperative preparation

A. Check latex allergy cart for supplies.

B. Call pharmacy and order all drugs you might need (dispensed in glass syringe).

C. Notify O.R. nurses on service. No latex gloves or latex products should come into contact with the patient. Neoprene (non-latex) gloves need to be obtained.

7. Anesthesia setup and care

A. Set up a regular circuit on the anesthesia machine and use a neoprene reservoir bag. Use plastic masks (adult or pediatric).

B. Draw up drugs in glass syringes from glass ampules. In an emergency, the rubber stoppers can be popped and drug drawn up in a glass syringe.

C. IV infusion setup with two three way stopcocks and no injection ports. (Alternatively tape all injection ports over and do not use).

D. Use Webril under the rubber tourniquet for IV placement. Teflon catheters can be used safely (e.g. angiocath). If BP cuff is rubber, use Webril under it.

E. Latex allergy should not alter your choice of anesthetic technique. There are no drugs that are specifically contraindicated.

8. Diagnosis of latex anaphylaxis

A. Anaphylaxis has been reported even in patients pre-treated with H_1, H_2 blockers and steroids and managed in a latex-free environment. Always be prepared to treat.

B. Onset is generally 20 - 60 minutes after exposure to the antigen.

C. Anaphylaxis presents with the clinical triad of hypotension (most common sign), rash, and bronchospasm.

D. Serum mast cell tryptase levels are high during an episode and up to 4 hours after. This test will help confirm the diagnosis of anaphylaxis, but do not identify latex as the antigen.

9. Treatment of latex anaphylaxis

A. Treatment of latex anaphylaxis does not differ from the treatment of other forms of anaphylactic reaction.

B. Primary treatment.

1. Stop administration of latex (usually gloves in contact with peritoneum).
2. Administer 100% oxygen.
3. Restore intravascular volume (2 - 4 litres of crystalloid).
4. Epinephrine, in adequate doses, is crucial for the successful treatment of anaphylaxis. The dose and route of epinephrine depend upon the severity of the reaction. Start with a dose of 10 ug, or 0.1 ug/kg and escalate rapidly to higher doses depending on the response.

C. Secondary treatment may include

1. Corticosteroids (0.25 - 1 g hydrocortisone or 1 - 2 g methyl-prednisolone).
2. Diphenhydramine 50-75 mg IV.
3. Aminophylline (5 - 6 mg/kg over 20 minutes for persistent bronchospasm).
4. Sodium bicarbonate (0.5 - 1 mEq/kg for persistent hypotension with acidosis).

Hematology and Anesthesia

1. **Factor VIII deficiency (hemophilia A)**
   A. The half life of factor VIII in plasma is 8-12 hours.
   B. Infusion of 1 unit of factor VIII per kg of body weight will increase the factor VIII activity level by 2%.
   C. Formula to calculate the required dose (to achieve a level of 100%)
      1. X number of units = weight in kg x (100 - present level)/2
      2. "X" number of units must be given initially, and half of "X" every 8-12 hours, to maintain the desired 50% level. Factor VIII may be obtained from fresh frozen plasma (1 unit of factor VIII/ml).
2. **Factor IX deficiency (hemophilia B; christmas disease)**
   A. The half life of factor IX in plasma is 24 hours.
   B. Infusion of 1 unit of factor IX per kg of body weight will increase the factor IX activity level by 1%.
3. **Common causes of a prolonged PT**
   A. Liver disease, ASA, vit K def, circulating anticoagulants, warfarin, dilutional coagulopathy, excessive heparin, DIC.
4. **Common causes of a prolonged PTT**
   A. Spurious results in heparin, delay in performing test, hemophilia, circulating anticoagulants, Von Willebrand's.
5. **Common drugs inhibiting platelet function**
   A. Aspirin, NSAIDs, alcohol, high dose penicillin or ticarcillin, heparin, moxalactam.

Coagulation Test Abnormalities

	PT	PTT	TT	Fibrinogen
Advanced Liver Disease	+	+	N or +	N or -
DIC	+	+	+	-
Vitamin K deficiency	++	+	N	N
Warfarin therapy	++	+	N	N
Heparin therapy	+	++	+	N
Hemophilia Factor VIII deficiency Factor IX deficiency	 N N	 + +	 N N	 N N
Factor VII deficiency	+	N	N	N
Factor XIII deficiency	N	N	N	N

Diagnosis of Coagulopathy

	PT	PTT	PC	BT	TT	CLT	SFM
Thrombo-cytopenia	Nl	Nl	Low	Nl or High	Nl	Nl	Nl
Hypofibrin-ogenemia	High	Nl or High	Nl	Nl	High	Nl	Nl or Pos
Clotting cascade deficit	High	High	Nl	Nl	Nl or High	Nl	Nl or Pos
Primary fibrinolysis	Nl	Nl	Nl	Nl	Nl	High	Pos
DIC		High	High	Low	High	Nl or High	High

PT=prothrombin time; PTT=partial thromboplastin time; PC=platelet count; BT=bleed time; TT=thrombin time; CLT=clot lysis time; SFM=soluble fibrin monomers.

Laboratory Tests to Assess Clotting Abnormalities

Abnormal Test	Indicate
Partial thromboplastin time	Intrinsic clotting cascade (XII, XI, IX, VIII)
Prothrombin time	Extrinsic clotting cascade (VII, X, V, II, fibrinogen)
Thrombin time	Fibrin formation
Plasma clot lysis time	Activity of fibrin degradation
Urea solubility	Fibrin cross linking (XIII)
Soluble fibrin monomers	Disseminated intravascular coagulation
Platelet count	Number of platelets
Bleeding time	Number and function of platelets

1. Coagulation tests

A. Partial thromboplastin time (PTT)

1. Partial thromboplastin is substituted for platelet phospholipid and eliminates platelet variability.
2. PTT measures the clotting ability of all factors in the intrinsic and common pathways except factor XIII.
3. Normal PTT is about

B. Activated partial thromboplastin time (aPTT)

1. An activator is added to the test tube before addition of partial thromboplastin added.
2. Maximal activation of the contact factors (XII and XI) eliminates the lengthy natural contact activation phase and results in more consistent and reproducible results.
3. Normal aPTT is 25-35 seconds.

C. Prothrombin time (PT)

1. Performed by measuring the time needed to form a clot when calcium and a tissue extract are added to plasma.
2. PT evaluates the extrinsic clotting mechanism and final common pathway, which includes Factors I, II, V, VII and X.
3. Normal PT is 10-12 seconds.

D. International normalized ratio (INR)

1. Developed to improve the consistency of oral anticoagulant therapy.
2. Converts the PT ratio to a value that would have been obtained using a standard PT method.
3. INR is calculated as $(Pt_{patient}/Pt_{normal})^{ISI}$ (ISI is the international sensitivity index assigned to the test system).

2. Thromboelastrogram

A. Thromboelastrography (TEG) is a method of testing for global assessment of coagulation. This technique uses a small sample of blood placed in a slowly rotating cuvette at 37°C. A piston is suspended in the cuvette, and as the coagulation proceeds, the tension on the piston is measured. A tracing is generated and several parameters are measured (see below).

B. Thromboelastrogram parameters

1. r (reaction time): start of recording until 1 mm deflection (represents initial fibrin formation).
2. k (clot formation time): measured from r until there is a 20 mm deflection in the tracing.
3. a (angle): slope of the increase from r time to k time.
4. MA: maximum amplitude in millimeters, a measure of the maximum clot strength (dependent on fibrinogen, level, platelet numbers, and function).
5. A^{60}: deflection measure at 60 minutes after MA (represents clot lysis and retraction).

3. Sonoclot

A. The sonoclot is a test of whole blood that utilizes a warmed cuvette and a suspended piston apparatus, This piston vibrates up and down very rapidly in the blood sample and Sonoclot detects any imedance to this vibration. As a result, the test follows the changes in viscosity over time.

4. Activated clotting time (ACT)

A. The ACT provides a global measurement of hemostatic function and is measured after whole blood is exposed to a specific activator of coagulation. The time for in vitro clot formation after whole blood is exposed to diatomaceous earth (celite) is defined as the ACT. Normal is 90 to 120 seconds. The linear increase in ACT seen with increasing doses of heparin provides a convenient method to monitor heparin's anticoagulant effect. Although the ACT test is simple, it lacks sensitivity to clotting abnormalities.

Sickle Cell

1. Sickle cell anemia is a hemoglobinopathy that results from inheritance of a gene for a structurally abnormal beta globin chain. This results in HbS which has two forms.

A. Sickle cell trait (HbAS): heterozygous state. Only 1% of the red cells in heterozygote's venous circulation are sickled. These patients are usually asymptomatic. Vigorous physical activity at high altitude, air travel in unpressurized planes, and anesthesia are potentially hazardous.

B. Sickle cell disease (HbSS): homozygous state: Seventy to 98%

2. Clinical features

A. Signs and symptoms include anemia (HgB levels 6.5 to 10 gm/dl), obstructive or hemolytic jaundice, joint and bone pain, abdominal and chest pains, lymphadenopathy, chronic leg ulcers, hamaturia, epistaxis,priapism, finger clubbing, and skeletal deformities.

B. The disease is characterized by periodic exaggeration of symptoms or sickle cell crisis. There are four main types of crises.

1. Vaso-occlusive crises: caused by sickled cells blocking the microvasculature, characterized by sudden onset of pain frequently with no clear-cut precipitating event.
2. Hemolytic crises: seen in patients with sickle cell disease plus G-PD deficiency, has hematologic features of sudden hemolysis.
3. Sequestration crises: involves sequestration of red blood cells in the liver and spleen causing their massive, sudden enlargement and an acute fall in peripheral hematocrit, this can progress to circulatory collapse.
4. Aplastic crises: characterized by transient episodes of bone marrow depression commonly occurring after viral infection.

Anesthesia and Chemotherapy Agents

1. Cyclophosphamide (Cytoxan): myelosuppression, hemorrhagic cystitis, water retention, pulmonary fibrosis, plasma cholinesterase inhibition.
2. Nitrogen Mustard: myelosuppression, local tissue damage.
3. Vincristine: neurotoxicity, dilutional hyponatremia.
4. Vinblastine: myelosuppresion.
5. Methotrexate: renal tubular injury.
6. 5-Fluorouracil and ARA C: hemorrhage enteritis, diarrhea, myelosuppression.
7. Adriamycin: cardiac toxicity; risk factors include total cumulative dose over 550 mg/m2, concomitant cyclophosphamide therapy, prior history of heart disease, age over 65 years.
8. Bleomycin: pulmonary toxicity; risk factors include total cumulative dose over 200 mg, concomitant thoracic radiation therapy, age over 65 years, increased oxygen concentration (?).
9. Mitomycin C: pulmonary toxicity.
10. Cisplatin: renal toxicity, neurotoxicity.
11. Nitrosoureas (BCNU, CCNU): myelosuppression, renal pulmonary toxicity.
12. Taxol: hypersensitivity reaction, myelosuppression, cardiac toxicity, peripheral neuropathy.
13. Growth factors: pulmonary edema, pericardial and pleural effusions

Anesthesia and Endocrinology

Diabetes Mellitus

1. Complications

A. Atherosclerosis: coronary and cerebral.
B. Microangiopathy: retinopathy and nephropathy.
C. Infections.
D. Decreased wound healing (tensile strength).
E. Peripheral neuropathy.
F. Autonomic dysfunction (orthostatic hypotension, resting tachycardia, early satiety, neurogenic bladder, impotence, delayed gastric emptying, painless myocardial ischemia).

2. Testing the degree of a patient's autonomic neuropathy

A. HR response to valsalva. The ratio of the longest R-R interval after the maneuver to the shortest R-R interval during the maneuver determined by ECG tracing. Abnormal is defined as a ratio of <1.10.
B. HR response to standing. The ratio of the R-R interval at the 30th beat after standing to the R-R interval at the 15th beat. Abnormal is defined as <1.00.
C. Beat to beat variability. The difference between the minimum and the maximum heart rate during periods of inspiration and expiration breathing at 6/min. Abnormal defined as a difference of <10 beat/min.
D. BP response to standing (orthostatic hypotension): abnormal is defined as a drop >30 mmHg.
E. Resting heart rate of >100.

3. Regimens for perioperative control

A. Nontight control regimen (for inpatients)

1. Aim: to avoid hyperglycemia, ketoacidosis and hyperosmolar states.
2. Day before surgery: NPO after midnight.
3. Day of surgery, start IV D5 1/2 NS (at maintenance rate).
4. After IV started, give 1/2 the usual AM insulin dose.
5. Continue D5 1/2 NS intraoperatively.
6. Check blood glucose at the start of surgery and every hour intraoperatively, treat with sliding scale.
7. Check glucose in PACU and treat with sliding scale.
8. Continue IV until patient taking and tolerating PO's.

B. Tight control regimen (for inpatients)

1. Overall aim is to keep blood glucose 80-200 mg/dl.
2. Evening before surgery, start IV D5W @ 50 ml/hr/70 kg.
3. Start regular insulin piggy back (50 U/250 cc NS = 0.2 units/cc); flush tubing with 60 cc before starting.
4. Insulin rate in U/hr = BS/150 (use 100 as denominator if patient is on steroids, e.g., 100 mg prednisone, or is markedly obese, or infected). Alternative dosing: 0.1 units/kg/hr.
5. Check blood glucose every 2-4 hrs and adjust insulin infusion to keep glucose levels between 100-200 mg/dl.
6. Day of surgery: continue D5W and insulin infusion (for patients undergoing major surgery consider rechecking plasma glucose and potassium on morning of surgery).

7. Check blood glucose at the start of surgery and every hour intraoperatively, adjust insulin infusion as needed.

4. Other medications and orders

A. One can estimate the soluble (regular) insulin requirement by taking the daily dose of lente units and multiplying by 1.5. This gives the units of regular insulin required per day.

B. One unit of insulin (IV) will lower the blood sugar by 30-40 mg/dl in a 70 kg person.

C. Ten grams of dextrose will raise the blood sugar by 30-40 mg/dl in an average 70 kg person.

D. Premeds: Zantac (50 mg IVPB) and metoclopramide (10 mg); (consider bicitra 30 cc).

5. Misc information

A. One in four insulin dependent diabetics may have "stiff joint syndrome" and may be difficult to intubate.

B. Diabetics may have gastroparesis as a result of autonomic neuropathy. The drug of choice for a nauseated or vomiting diabetic is metoclopramide 10 mg IV.

C. In obstetrics, the insulin requirements may drop dramatically after delivery.

D. Lactate is converted to glucose in the liver. Lactated Ringers may cause a blood glucose elevation.

Types of Insulin

	Insulin Type	**Onset**	**Peak Action**	**Duration**
Short	Regular	15-30 min	1-3 hours	5-7 hours
	Semilente	30-60 min	4-6 hours	12-16 hours
Intermediate	Lente, NPH	2-4 hours	8-10 hours	18-24 hours
Long	Ultralente	4-5 hours	8-14 hours	24-36 hours

Oral Hypoglycemic Agents

Drug	Onset (hrs)	Duration (hrs)
Tolbutamide (Orinase)	0.5-1	6-12
Acetohexamide (Dymelor)	0.5-1	12-24
Tolazamide (Tolinase)	4-6	10-18
Chlorpropamide (Diabinese)	24-72	60-90
Glyburide (Micronase)	0.5-1	24-60
Glipizide (Glucotrol)	1-3	12-24

Pheochromocytoma

1. **Definition:** catechol-secreting tumors derived from chromaffin tissue (usually the adrenal medulla, but may occur in a variety of other locations). Most pheochromocytomas produce both norepinephrine and epinephrine. Pheochromocytomas may be associated with multiple endocrine neoplasia syndromes, types I and II. Endogenous catecholamine levels should return to normal levels within 1-3 days after successful removal of the tumor. Overall mortality: 0-6%.
2. **Clinical manifestations**
   A. Triad of symptoms: palpitations, headaches, and diaphoresis.
   B. Signs: paroxysmal hypertension (hallmark).
   C. Less common clinical manifestations, abdominal pain, chest pain, GI symptoms, weakness, visual symptoms.
3. **Diagnosis:** confirmed by abnormally high levels of catecholamines or catechol metabolites in blood or urine. Assay of urinary metanephrine remains the most reliable indicator of excess catecholamine secretion.
4. **Preoperative evaluations**
   A. Prazosin or phenoxybenzamine may be used to produce preoperative alpha-adrenergic blockade. Ten to 14 days is usually required for adequate alpha-receptor blockade as evidenced by orthostatic hypotension, nasal stuffiness, and decrease sweating. Beta blockade is instituted after the onset of adequate alpha blockade if dysrhythmias or tachycardia persists.
   B. Preoperative goals include blood pressure below 160/95, no ST-T wave changes, and maximum of one PVC per 5 minutes.
5. **Anesthetic considerations**
   A. Overall goal is to avoid sympathetic outflow.
   B. Drugs to avoid:
      1. Histamine releasers: morphine, curare, atracurium, etc.
      2. Vagolytics and sympathomimetics: atropine, pancuronium, gallamine, succinylcholine.
      3. Myocardial sensitizers: halothane.
      4. Indirect catechol stimulators: droperidol, ephedrine, TCA's, chlorpromazine, glucagon, metoclopramide.
   C. Monitors: intraartial catheter in addition to standard monitors.

Hyperthyroidism

1. **Clinical manifestations may include:** weight loss, heat intolerance, muscle weakness, diarrhea, hyperactive reflexes, nervousness, exophthalmos, sinus tachycardia/atrial fibrillation and fine tremors.
2. **Management of anesthesia**
   A. Preoperative: all elective surgery should be postponed until the patient is rendered euthyroid with medical treatment. Preoperative assessment should include normal thyroid function tests, and a resting heart rate less than 85-90 beats/min. The combined use of beta antagonists and potassium iodide is effective in rendering most patients euthyroid in 10 days.
   B. Intraoperative: thiopental is the induction agent of choice, since it possesses some antithyroid activity. Drugs that will stimulate the sympathetic nervous system should be avoided (ketamine, pancuronium, indirect-acting adrenergic agonists, etc.). MAC requirements for inhaled agents or anesthetic requirements are not increased with hyperthyroidism. Cardiovascular function and body temperature should be closely monitored.
   C. Postoperative: most serious postoperative problem is thyroid storm, which is characterized by hyperpyrexia, tachycardia, altered consciousness, and hypotension. Most commonly occurs 6-24 hours postoperatively. Treatment includes hydration and cooling; propranolol (0.5 mg increments until heart rate is below 100 beats/min); propylthiouracil (250 mg every 6 hours orally) followed by sodium iodide (1 gm IV over 12 hours); and correction of any precipitating cause.
   D. Complications after total or partial thyroidectomy: recurrent laryngeal nerve palsy, hematoma formation, hypothyroidism, and hypoparathyroidism.

Hypothyroidism

1. **Clinical manifestations may include:** generalized reduction in metabolic activity, lethargy, intolerance to cold, weight gain, constipation and decreased cardiac function.
2. **Myxedema coma** results from extreme hypothyroidism and is characterized by impaired mentation, hypoventilation, hypothermia, hyponatremia, and congested heart failure. Treatment is with IV thyroid hormones (300-500 micrograms of levothyroxine sodium in patients without heart disease).
3. **Management of anesthesia**
   A. Preoperative: patients with uncorrected severe hypothyroidism or myxedema coma should not undergo elective surgery. Mild to moderate hypothyroidism is not a absolute contraindication to surgery. Patients should be treated with histamine H_2 blockers and metoclopramide because of their slowed gastric emptying times.
   B. Intraoperative: ketamine is the induction agent of choice because of the exquisite sensitivity of hypothyroid patients to drug-induced myocardial depression. MAC requirements for inhaled agents are not changed with hypothyroidism.
   C. Postoperative: recovery from general anesthesia may be delayed by slowed drug biotransformation, hypothermia, and respiratory depression.

Obesity

1. Definitions

- **A. Overweight:** up to 20% more than predicted ideal body weight.
- **B. Obesity:** more than 20% above ideal body weight.
- **C. Morbid obesity:** more than twice as much as their ideal body wt.

2. Body mass index

- **A.** Clinically the most useful index for defining obesity.
- **B. Body mass index** (BMI) = weight (kg)/height^2 (meters squared)
- **C.** A BMI greater then 28 kg/m^2 defines obesity; BMI greater than 35 kg/m^2 defines morbid obesity.

3. Clinical manifestations

- **A. Cardiovascular:** systemic hypertension, cardiomegaly, congestive heart failure, coronary artery disease, pulmonary hypertension. Cardiac output increases approximately 0.1 L/min/kg of adipose tissue primarily through increases in stroke volume (as opposed to heart rate).
- **B. Ventilation:** decreased lung volumes and capacities (suggestive of restrictive lung disease), arterial hypoxemia (decreased functional residual volume predisposes the obese patient to a rapid decrease in PaO_2), obesity-hypoventilation syndrome, decreased chest wall compliance (pulmonary compliance is normal).
- **C. Liver:** abnormal liver function tests, fatty liver infiltration.
- **D. Metabolic:** insulin resistance (diabetes mellitus), hypercholesterolemia.
- **E. Gastrointestinal:** hiatal hernia, gastroesophageal reflux, poor gastric emptying, hyperacidic gastric fluid.

4. Obesity-hypoventilation syndrome (Pickwickian syndrome)

- **A.** Occurs in approximately 8% of obese patients, most commonly in the extremely obese.
- **B.** Obstructive sleep apnea consists of absent nasal and oral airflow during sleep despite continuing respiratory effort. This is generally due to backward tongue movement and pharyngeal wall collapse (glossoptosis) secondary to interference with the normal coordinated contraction of pharyngeal and hypopharyngeal muscles.
- **C.** Characterized by hypercapnia, cyanosis-induced polycythemia, right-sided heart failure, and somnolence. The presence of episodic daytime somnolence and hypoventilation in a morbidly obese patient suggests the presence of this syndrome.
- **D**. Obstructive sleep apnea is diagnosed by finding at least 30 episodes of apnea (of duration at least 10 seconds) in a 7-hour study period.

5. Anesthetic considerations

- **A. Preoperative**
  1. Preoperative evaluation of morbidly obese patients undergoing major surgery should include chest x-ray, EKG, arterial blood gas, and pulmonary function tests.
  2. The airway should be carefully examined, since these patients are often difficult to intubate as a result of limited mobility of the temporomandibular and atlanto-occipital joints, a narrowed upper airway, and a shortened distance between the mandible and sternal fat pads.
  3. All obese patients are at an increased risk of developing aspiration pneumonitis and should be considered full-

stomachs. Pretreatment with H_2 antagonists (both the night before and the morning of surgery), metoclopramide, and sodium citrate should be considered.

B. Intraoperative

1. The risk of rapid decreases in PaO_2 emphasizes the importance of preoxygenation prior to intubation.
2. Rapid sequence induction/intubation is usually selected to minimize the risk of pulmonary aspiration. Morbidly obese patients with a difficult airway should be intubated awake.
3. Volatile anesthetics may be metabolized more extensively in obese patients. Obese patients are at an increased risk of halothane hepatitis.
4. Obese patients generally require 20-25% less local anesthetic for spinal or epidural anesthesia secondary to epidural fat and distended epidural veins.

C. Postoperative

1. Respiratory failure is the major postoperative problem of morbidly obese patients. Other problems include deep vein thrombosis, pulmonary embolism, and wound infections.
2. The semisitting position will optimize the mechanics of breathing (unload the diaphragm) and to minimize the development of arterial hypoxemia.
3. Obese patients generally should not be extubated until fully awake.

Anesthesia and Liver Disease

1. **Laboratory test for prediction of outcome in cirrhotics**
   - **A. Albumin:** indicates liver synthetic function and may correlate with wound healing. Half life is 26 days. Low levels reflect chronicity of disease.
   - **B. Prothrombin time (PT):** relates to clotting factor synthesis, and since half-time is relatively short, assesses liver function on an hour-to-hour basis. Prolonged values reflect severity of disease.
   - **C. Bilirubin:** assesses overall state of hepatic function.
2. **Preoperative evaluation and treatment**
   - **A. Past medical history:** evaluation of type of liver disease, previous or present jaundice, history of blood transfusions and gastrointestinal bleeding; previous surgical operations, anesthetic management; degree of either ascites or encephalopathy should be noted.
   - **B. Lab test:** CBC with platelet count, serum bilirubin, albumin, serum electrolytes (particularly sodium, potassium, and glucose), creatinine and BUN, ABG, PT/PTT and liver function tests.
   - **C. Preop LFT's:** Transaminases should be stable or decreasing for elective surgery.
   - **D. Treatment:** the coagulation system function should be evaluated and treated preoperatively (with vit K, FFP, or platelets as needed). Adequate hydration and diuresis (approximately 1 ml/kg/hr) should be achieved preoperatively.
   - **E. Premedications:** all medications needed to control the diseased state should be administered. Sedatives should be omitted or the dose decreased.
3. **Algorithms for elevated liver function tests**
   - **A. Algorithm for elevated serum transaminase**
     1. High level >500 are almost always hepatic in origin.
     2. A gamma-glutamyl transpeptidase may assist in pin pointing hepatic etiology.
     3. Alcoholic hepatitis presents with an AST/ALT ratio >2:1.
     4. Suspect tissue trauma if patient is asymptomatic patient and serum is only mildly elevated (<60 to 80).
   - **B. Algorithm for elevated serum alkaline phosphatase**
     1. If this is the only abnormality found, it may not be hepatic in origin.
     2. Rule out primary biliary cirrhosis. Rule out drugs used.
   - **C. Algorithm for elevated serum bilirubin:**
     1. Rule out Gilbert's syndrome.
     2. If conjugated level is high, the cause is often hepatic.
     3. Bilirubin levels out of proportion to elevations of other liver function tests often due secondary to cholestasis or sepsis.

4. Associated complications in liver disease

A. Hyperkinetic cardiovascular system.
B. Dysrhythmias.
C. Hypoxia related to venous admixture.
D. Increased intraabdominal pressure.
E. Susceptibility to renal failure.
F. Susceptibility to infection.
G. Poor temperature control.
H. Anemia.
I. Coagulopathy.
J. Electrolyte imbalance.

5. Liver transplantation

A. Common indications for liver transplantation

1. Fulminant hepatic failure (viral or drug), Reye's syndrome, massive trauma, cirrhosis, sclerosing cholangitis, Budd-Chiari syndrome, hepatocellular carcinoma.

B. Absolute contraindications to liver transplantation

1. Extrahepatic hepatic malignancy.
2. Active sepsis outside the hepatobiliary tree.
3. Severe cardiopulmonary disease.
4. AIDS.
5. Thrombosis of the portal and superior mesenteric veins.

C. Relative contraindications to liver transplantation

1. Age greater than 60 years.
2. Hypoxemia related to intrapulmonary shunts.
3. HIV positive (without clinical AIDS).
4. HBsAg positive.
5. Prior complex hepatobiliary surgery.
6. Active alcoholism or drug abuse.
7. Inability to understand the overall procedure.

Overview of the Orthotopic Liver Transplantation Procedure

Phase	Surgical Procedures	Physiologic Changes
Preanhepatic	Dissection of porta hepatitis Release of hepatic attachments	Third space loses (ascites) Hemorrhage (venous collaterals)
Anhepatic	Clamp hepatic artery and portal vein Venovenous bypass Clamp IVC	Obstruction of venous return; Oliguria Atelectasis, decreased compliance
Neohepatic	Anastomosis of IVC Flush hepatic allograft Anastomosis of portal vein and hepatic artery Biliary drainage procedure	Hemorrhage (coagulopathy) Hyperkalemia Hypothermia Metabolic acidosis

Classification of Hepatic Reserve and Surgical Risks

	Group A	Group B	Group C
Serum Bilirubin (mg/dl)	<2.0	2.0-3.0	>3.0
Serum Albumin	>3.5	3.0-3.5	<3.0
Ascites	None	Controlled	Poorly controlled
Neurologic Status (encephalopathy)	None	Minimal	Advanced
Nutritional Status	Excellent	Good	Poor
Prothrombin Time (sec above)	35,067	35,160	>6
Surgical Risk	Good	Moderate	oor
Mortality Risk	<5%	25%	>50%

Critical Care Medicine

1. Nutritional Requirements

Non-protein calories (cal/kg/day)

	Carbohydrates and lipids	Protein (gm/kg/day)
Normal	25	1.0
Multiple Trauma	25-35	1.5-2.5
Sepsis	30-40	1.5-2.5
Burn	25-50	1.5-3.0

Optimal ratio of 100-200 calories per gram of nitrogen intake

2. Basal energy expenditure/requirements (Harris Benedict Equation)

A. Male = 66 + (13.7 x wt , kg) + (5 x Height, cm) - (6.8 x Age, yrs).

B. Female = 65.5 + (9.6 x wt , kg) + (1.8 x Height, cm) - (4.7 x Age, years).

3. Calorie count

A. D_{10}, 1 liter = 340 calories

B. D_{25}, 1 liter = 850 calories

C. Intralipid 10%, 500 cc = 500 calories

D. Intralipid 20%, 500 cc = 1000 calories

E. Amino 10%, 1 liter = 340 calories

4. Nutritional assessment

A. Albumin: >3 gms/dl adequate (better to check pre-albumin levels).

B. Nitrogen Balance: protein intake (grams)/6.25 - Urine Nitro - 4

1. Urine Nitro = total gms of nitrogen in 24 hr urine collection.

C. Total lymphocyte Count: <1000 - 1500/microliter, moderate to severe malnutrition.

D. Transferrin: <100 - 200, moderate to severe malnutrition.

E. Prealbumin level.

F. Daily weights.

5. Oliguria

	Prerenal	**Hepatorenal**	**Renal (ATN)**
U osm	>500	>100	<350
U Na	<10-20	<10-20	>20-40
U/Pl creatinine ratio	>20:1	>30:1	<20:1
Fractional Excretion of Na	<1	<1	>1
BUN/Cr ratio	>20:1		<10:1
Urine Specific Gravity	>1.015		<1.015

Fractional Excretion of Na =[(Urine Na) x (Plasma Cr)]/[(Plasma Na) x (Urine Cr) x 100.]

Creatinine Clearance = (Urine Cr in 24 hours)/14.4 x (Plasma Cr).

6. Adult respiratory distress syndrome (ARDS)

A. Clinical setting.

B. Exclusion of underlying cardiac and chronic pulmonary disease.

C. Respiratory distress: dyspnea; increased respiratory rate (>35).

D. Laboratory findings

1. Arterial oxygen tension <50 mmHg on 60%.
2. Ventilation-perfusion mismatching: increased venous admixture and increased dead space ventilation.
3. Decreased lung compliance to <50 ml/cm H_2O.

E. CXR findings: early interstitial edema followed by alveolar edema.

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